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THE NATURE OF THE DECLINE IN THE LOSS OF INFANT AND FŒTAL LIFE: A STUDY IN WESTERN AUSTRALIAN VITAL STATISTICS.[1]

By J. C. EDWARDS,

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THE white population of Western Australia can congratulate itself and its infant health sisters that its infant mortality rate is among the lowest rates on record. Throughout the world statistics relating to infant mortality were bettered in 1948 only by Victoria and New Zealand. It must be remembered that the statistics in Western Australia include the native population and that Victoria has relatively fewer natives, while New Zealand excludes Maoris from these statistics (Commissioner of Public Health, Western Australia, 1948).

If, then, Western Australia is amongst those States leading the world in the reduction of infant mortality, it is of great interest to study in what ways this progress has been achieved and to attempt to estimate in what ways the rate may be reduced even further.

An examination of the causes of infant death over the last thirty-five years shows clearly where the source of improvement lies.

The relatively large part played by four specific causes of infant mortality may be demonstrated by statistics for 1948. In this year they caused 249 out of 331 infant deaths. It may be wondered that the infant mortality from respiratory diseases has not fallen decisively since the introduction of the sulphonamides. There are probably two factors preventing this favourable occurrence. The first is that the sulphonamides and the antibiotics do not have the same dramatic effect on pneumonia occurring in infants as is seen when the disease appears as lobar pneumonia in adults. Also there are in the southern parts of the State native children who have in winter insufficient protection from the cold and damp, and whose mothers do not seek or obtain for them when they are ill any medical treatment at a sufficiently early stage in the disease. There is a decisive but moderate fall in the infant deaths from prematurity and immaturity. I believe that the decline in deaths from these causes, as the decline in maternal eclampsia, is due to improved and more extensive antenatal care. It is not possible to do more than express this as an opinion. Except that we know that rubella may be a factor contributing to malformation, and that insulin can cause malformation experimentally, this category remains as much beyond our knowledge and control as ever.

The improvement lies almost wholly in the removal of gastro-enteritis as a cause of infant death. There is a slight reduction in the number of deaths from prematurity.

[1] This paper and the accompanying paper by D. J. R. Snow and A. T. Pearson are preliminary reports written in order to demonstrate to those interested in matters of public health the survey which is being attempted in Western Australia. The author of the present paper (J.C.E.) was in charge of the analysis of the stillbirth notifications received from the beginning of 1948 until the end of July, 1949. At this time post-mortem examinations were not being conducted as a routine procedure upon stillborn infants. From August 1, 1948, this work was in charge of the authors of the second paper (D.J.R.S. and A.T.P.), and post-mortem examinations were being conducted regularly. For this reason the earlier notifications have been analysed from a purely clinical point of view, and an attempt is made also to give a picture of the present state of infant health in Western Australia. The analysis of the latter notifications has been coordinated with the pathological findings. It is hoped at a later date, when the statistics are more adequate, to present a complete analysis of Western Australian stillbirths which correlates clinical and post-mortem findings.

but little improvement in the statistics of death from respiratory conditions and malformations. During these years there has been a considerable difference in the infant mortality rates recorded in town and country. In the twenties the infant mortality in the metropolitan area of Perth was much higher than in the country. In the forties this tendency was reversed, and the country rate was higher than the metropolitan. This change we should presumably associate with the improvement of sewerage

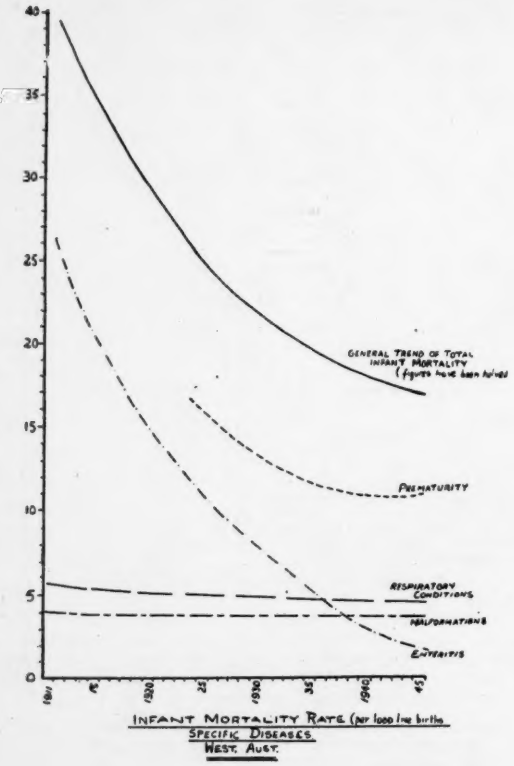

FIGURE I.

Infant mortality rate per 1000 live births; specific diseases, Western Australia.

and the banishment of the horse from the streets of the city, and with an improvement in the public understanding of the principles of hygiene and infant health. Certainly the disparity between metropolitan and country districts is mainly due to the bowel and respiratory infections.

It seems, therefore, that the reduction in infant mortality so far attained is derived from an elimination of gastro-enteritis, to an extent which is so great that we cannot expect any further considerable reduction in infant mortality from this source. In fact, infant mortality has become more and more related to the obstetrical and prenatal causes of death, and these causes are in many cases also the cause of stillbirths. Therefore it becomes advantageous to study stillbirth in conjunction with neonatal mortality. The infectious diseases which cause infant mortality are principally associated with the last eleven months of the first year of life, and the obstetrical and prenatal causes with the first month of life; hence the advantage of presenting statistics as is done in Table IV. Infectious diseases cause only 20% of neonatal mortality (death under one month of age); this is taken from "Neonatal Mortality and Morbidity" (published in 1949 by His Majesty's Stationery Office).

From Table I it will be seen that the greatest reduction has occurred in the postnatal rates, as would be expected. There has been a reduction in the stillbirth rates, but none in the number of deaths occurring within the first week of life. This is probably due to a "shunting" phenomenon—that is, a certain number of infants have been saved from stillbirth only to die within the first week of life, and so on. For 1948 the statistics can be presented as in Table III. It is a great achievement that in the metropolitan area only 2·2 infants per 1000 died from postnatal disease.

TABLE I.

Stillbirth and Infant Mortality Rates per 1000 Births.

Year.	Total Births, including Still-births.	Still-birth Rates.	Neonatal Rates.		Total Mortality Rates Under 1 Year.	Other Post-Natal Rates Over 1 Month and under 1 Year.
			Under 1 Week.	Under 1 Month.		
1926 ..	8,534	27·4	—	27·6	48·0	20·4
1927 ..	8,708	26·0	—	23·0	44·7	21·7
1928 ..	8,981	30·9	—	23·1	35·5	12·4
1929 ..	9,316	28·4	18·8	25·8	54·6	28·8
1930 ..	9,456	27·0	18·0	23·5	46·5	23·0
1931 ..	8,777	26·0	20·1	26·6	40·5	13·9
1932 ..	8,175	25·7	21·02	25·2	43·5	18·3
1933 ..	8,105	29·4	18·1	22·5	35·8	13·3
1934 ..	8,029	29·2	19·3	24·8	38·8	14·0
1935 ..	8,377	30·8	20·6	24·8	39·0	14·2
1936 ..	8,730	28·9	19·6	24·8	41·0	16·2
1937 ..	8,850	27·2	16·8	21·2	36·5	15·3
1938 ..	9,325	23·9	16·6	19·1	33·1	14·0
1939 ..	9,249	23·0	16·5	19·7	40·0	20·3
1940 ..	9,363	25·9	20·5	24·9	43·0	18·1
1941 ..	10,375	24·8	15·1	18·1	34·4	15·7
1942 ..	10,109	20·6	17·1	20·3	36·2	15·9
1943 ..	10,759	25·8	17·1	21·0	31·8	10·8
1944 ..	11,144	24·8	18·6	21·0	32·0	11·0
1945 ..	10,896	20·6	18·0	20·0	28·9	8·9
1946 ..	12,398	23·6	17·1	20·6	30·3	9·6
1947 ..	13,178	23·0	16·9	19·4	30·2	13·2
1948 ..	13,197	20·1	16·9	18·7	25·0	8·4

Stillbirths and infant deaths due to prenatal and obstetrical causes in 1948 have been tabulated in Table III from statistics taken from information supplied by the Registrar-General.

A further analysis made from notifications to the Department of Public Health suggests that toxæmia of pregnancy would be associated with 135 deaths.

TABLE II.

Death Rates per 1000 Live Births from	Metropolitan Districts.	Country Districts.
Prenatal causes	17·2	17·4
Natal and neonatal causes	3·1	4·3
Postnatal causes	2·2	7·1
Total (1948)	22·5	28·8

In 1948 the Western Australian Parliament amended the *Health Act* to provide for (*a*) the compulsory reporting of all stillbirths and premature births and the return of prescribed clinical information for research purposes, (*b*) compulsory autopsy on stillborn infants.

As a result of this Act and of the enthusiasm of the Commissioner of Public Health (then Dr. C. E. Cook), much clinical information was collected by the Department of Public Health, and this has been the basis of two other reports ("The Annual Report of the Commissioner of Public Health", 1949, Perth, Appendix VI; Snow and Pearson, 1951).

According to a definition prescribed under the *Health Act*, a stillbirth means the birth of any child not born alive, whose body weighs over two pounds twelve ounces—a weight which corresponds to twenty-eight weeks of

gestation. Hence these fœtuses have survived twenty-eight weeks of intrauterine life, but have died prenatally or from causes arising out of parturition itself. I have analysed notifications received at the department between January, 1948, and July 31, 1949. In the examination of this clinical information the guiding principle has been not so much to present scientific and accurate statistics as to attempt to estimate in each individual instance—often with incompletely detailed information—the fundamental abnormal state or procedure associated with the infant's death. Thus, the purpose of the survey is to provide a basic knowledge upon which recommendations may be made, which will lower the number of stillbirths and obstetrical infant deaths in Western Australia. Details of this information are given in Table IV.

TABLE III.

Specific Causes of Infant Deaths (1948).	Still-births.	Infant Deaths.	Total Deaths.
Congenital malformations	11	63	73
Congenital syphilis	—	2	2
Hæmolytic diseases	10	17	27
Prenatal death or unspecified stillbirths	167	—	308
Prematurity	—	141	
Placenta prævia	5	—	5
Toxæmia	15	—	15
Accidental hæmorrhage	22	1	23
Asphyxia, birth fatalities and injuries	35	40	75
Other causes	1	2	3
Total deaths	266	266	532

Stillbirths to Primiparæ and Multiparæ.

From figures given in the "Statistical Register" for the year 1945 it is calculated that four births in eleven are to *primiparæ* in Western Australia. A division of stillbirths into those to *primiparæ* and those to *multiparæ* shows that the stillbirth ratio in the former category is 99:100 of the *multiparæ* rate. When the same distinction is drawn between breech births only, it is found that the rate for *primiparæ* is 117:100 of the rate for *multiparæ*. This emphasizes the special attention which it is necessary to give these *primiparæ*; such breech births should be prevented when possible by antenatal external version, especially as breech births to *primiparæ* are often complicated by extension of the infant's legs.

Stillbirth following Cephalic and "Breech" Delivery.

In 1948, out of 266 stillbirths registered with the Registrar-General 139 were notified to the Department of Public Health. The relative risks of cephalic and breech delivery can be studied by the assumption that in 1948 the number of breech births (15) amongst the notified stillbirths (139) is a fair sample of the number of the breech stillbirths among the registered stillbirths (266). If this is so, there should have been 29 stillbirths following breech delivery during 1948 in Western Australia. As has been previously remarked, in all cases marked as breech stillbirths there was no information suggesting a more fundamental cause of death (for example, disproportion, malformation, rhesus factor incompatibility, prenatal death *et cetera*). It is generally accepted that approximately one pregnancy in 30 terminates as a breech delivery. Therefore, in 1948 there should have been approximately 440 breech births. If this is so, then 6·6% of breech births terminated in stillbirth, for no other apparent reason than difficulty or injury at delivery. When similar calculations are applied to cephalic version, forceps delivery, cord constriction, and disproportion, it is found that this group accounts for 70·1 deaths out of 12,757 live births and stillbirths. This calculation gives a mortality rate of 0·55% for cephalic delivery as a risk in itself. The risks of being born by the breech are approximately twelve times as great as being born by the vertex. If in all breech presentations external version without anæsthesia was performed at about the thirty-fourth week of pregnancy, it is believed that many of these breech stillbirths could be avoided. There should be very few deaths from placental separation if external version is performed then, as it is a simple procedure at this stage.

These facts emphasize a point at which antenatal care can assist in lowering the stillbirth rate, and the importance of a routine antenatal examination between the thirty-second and thirty-fourth weeks of pregnancy. Any breech presentation persisting after the thirty-fourth week should be corrected or, failing this, further investigations should be undertaken to determine the possibility of any other coexisting and causative abnormality. The basis of the antenatal examination is the testing of the urine, the reading of the blood pressure and the palpation of the *fœtus in utero*. All these are procedures within the scope of a midwife's training. Patients' clinical histories should be recorded upon adequate antenatal charts. All patients should be examined at least fortnightly after the thirty-second week of pregnancy.

TABLE IV.

Total Stillbirths, January, 1948, to July, 1949: Stillbirths 219, Toxæmia 54.[1]

Group.	Cause.	Stillbirths.		
		Total.	To *Primiparæ.*	To *Multiparæ.*
Prenatal death 96 (Toxæmia 17[1])	Interference	3	3	—
	Malformation	14	9	5
	Syphilis	1	—	1
	Rh incompatibility	9	9	—
	Toxæmia	15	7	8
	Other causes	14	11	3
	Unknown	40	21	19
Death during labour (or immediately preceding) 39 (Toxæmia 20[1])	*Placenta prævia*	9	7	2
	Accidental hæmorrhage	17	13	4
	Toxæmia	3	2	1
	Uterine inertia	6	2	4
	Unknown	4	3	1
Death at delivery 84 (Toxæmia 17[1])	Breech presentation	25	15	10
	Forceps delivery	13	4	9
	Cord constriction	10	7	3
	Normal delivery	13	11	2
	Toxæmia	11	9	2
	Disproportion	12	7	5

[1] Wherever figures are given for toxæmia, they represent the number of cases in that group complicated by toxæmia of pregnancy. Of the last three columns in the table, the first includes all stillbirths, the second stillbirths to *primiparæ*, and the third stillbirths to *multiparæ*

Forceps Delivery.

In these statistics normal unaided cephalic deliveries designated as normal delivery and forceps delivery were followed by stillbirth in 13 cases each. There are only two cases notified which are reported as "failed forceps". Under the heading of disproportion, in almost all the cases delivery was by forceps or by the breech. Such cases are not included under the headings discussed in this paragraph. In many cases in which forceps delivery was required, occipito-posterior presentations required correction.

Toxæmia of Pregnancy.

It will be noted that 25·02% of stillbirths were complicated by maternal toxæmia. This group includes all patients whose clinical history showed albuminuria, who had a blood pressure elevated above 140 millimetres of mercury, systolic, and 90 millimetres, diastolic, who gave birth to a macerated fœtus after having an attack of eclampsia in a previous pregnancy, or whose pregnancy was terminated by an accidental hæmorrhage (*ablatio placentæ*). This percentage would correspond to 67 cases among the registered stillbirths.

Toxæmia is accepted to be a common cause of prematurity ("Neo-natal Mortality and Morbidity", 1949). The placenta is apparently an important factor in the main-

tenance of pregnancy by warding off the onset of labour. Degeneration and infarction of the placenta occur more rapidly under the influence of toxæmia and labour sets in prematurely, while the fœtus may already have died *in utero* for lack of oxygen or nourishment. Premature babies are particularly apt to suffer from intracranial hæmorrhage and asphyxia, because, although smaller, their tissues are far less able than those of full-term fœtuses to withstand the stresses imposed upon them during labour. Once born, they are still exceptionally vulnerable to infection and malnutrition through feeding difficulties, while their heat regulation is poor and their respiration lacks vigour.

For these reasons it is apparent that if it is possible by prophylactic measures to reduce the incidence of toxæmia of pregnancy, many infant lives will be saved per year. It is often stated in text-books on obstetrics that, although antenatal care has been established for the last thirty years, the results are not so good as was originally hoped. It is probable that this is due to the fact that, although antenatal care is practised, full advantage is not taken of its opportunities. The great part played by toxæmia of pregnancy in the causation of infant death has been amply demonstrated.

I hold views about the nature of the ætiology of toxæmia of pregnancy which I believe will not be generally accepted until the action of the adrenal hormones, A.C.T.H., and the sex hormones are better understood than they are today (Edwards, 1950). However, there is little dispute among obstetricians regarding the treatment of pre-eclampsia. The principles of treating this condition are as follows: (i) rest in bed; (ii) removal of the sodium ion from the diet and drugs consumed by the patient;[1] (iii) termination of gestation if the previous two measures fail.

Because the preeclamptic patient does not feel unwell in the early stages of the syndrome, antenatal examinations are of especial importance. To save infant life toxæmia must be recognized in its incipient stages and treated prophylactically. Furthermore, the first important sign is often not albuminuria, hyperpiesis or œdema, but it may be an abnormal increase in the weight of the patient. Therefore it is the study of the average increase in weight in pregnancy that requires far more attention than it has so far received. It is necessary that all women be rested in bed in the earliest stages of toxæmia. Such patients tend to lose weight rapidly and are relieved of the hyperpiesis.

This treatment is a social problem. A pregnant woman who feels well may be willing to accept the advice of her physician to go home and rest in bed, but often she will not be able in practice to follow the advice adequately. Therefore these patients, although they require little nursing care, require to be provided with a hospital bed for up to six weeks before confinement. Special beds should be established for such cases, and all practitioners should be encouraged to make use of them for their patients as soon as they show the early signs of toxæmia. It is believed that, if this is done, eclampsia can be shown to be a preventible disease. Many infant deaths will also be avoided by this means and much postnatal maternal morbidity avoided.

The Care of Premature Infants.

The care of premature infants is well understood and it is essentially a nursing problem. Every large centre should have a "premature" station, equipped with a special cot, in which temperature and humidity can be controlled and oxygen and carbon dioxide supplied. Breast milk should always be available for premature infants. The nursing staff of the station should be especially trained in this work. Suitable means of transport should be available to convey all premature infants in the district to this station.

[1] Recent work by Dieckmann *et alii* suggests that the elimination of electrolyte and water can be accelerated by diuresis if the patient is ordered to ingest 150 to 200 millilitres of water per hour (Dieckmann *et alii*, 1949.)

Lowering of the Stillbirth Rate.

It may be asked to what extent it may be possible to lower the present stillbirth rate of 20·5 per 1000 live births. In Britain (Baird, 1945, in "Neo-natal Mortality and Morbidity") statistics taken from private hospitals, whose patients belong to the higher income groups, show a stillbirth rate of 10·1 per 1000 births and a neonatal mortality of 8·1 per 1000 live births. It is probable that this approaches the limits which it is possible to reach in the light of present medical knowledge and skill.

Summary.

The decrease in infant mortality is shown to be due to the elimination of infantile gastro-enteritis. This leaves, as a hard core, the problem of reducing infant mortality from obstetrical and prenatal causes. These deaths tend to share their ætiology with that of stillbirth. It is suggested that the loss of infant life could be further reduced if a general policy was adopted of (i) correcting all breech presentations at the thirty-fourth week of gestation, and (ii) treating all patients showing the early signs of toxæmia of pregnancy by bed rest.

Acknowledgement.

I am indebted to Dr. Linley Henzell, Commissioner of Public Health for Western Australia, for permission to publish this article.

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STILLBIRTHS, NEO-NATAL DEATHS, AND PREMATURE BIRTHS IN WESTERN AUSTRALIA.

By D. J. R. SNOW and A. T. PEARSON,

Perth.

IN April, 1947, Dr. Cecil Cook submitted a blunt, vigorous and authoritative report to the Minister for Health in Western Australia on population trends in relation to health administration. In characteristic language he directed attention to "the progressive ageing of the existing population" and emphasized the need "to prevent the infant wastage associated with abortion, stillbirth, infant mortality, and chronic invalidity following birth injury".

He urged the appointment of an advisory committee on infant mortality (consisting of an obstetrician, physician, pædiatrician, pathologist, serologist, and midwife), for the following purposes: (i) investigating the causes of sterility, abortion, stillbirth, and neonatal trauma; (ii) preparing an ordered plan for medical and pathological research into the problems arising from its studies; (iii) recommending measures to be taken by statute and otherwise for the purpose of assisting its researches and implementing prophylactic measures. He finally suggested that "autopsy upon stillbirths in the metropolitan area should be made obligatory by law".

Ministerial approval for the constitution of this committee was secured on May 14, 1947, and its first meeting took place soon afterwards. A period of over two years elapsed, however, before the machinery for a suitable inquiry could be set in motion, and this report is an endeavour to present the preliminary findings arising out of the first 457 cases investigated.

TABLE I.

Presumptive Cause of Death.	Year.	Autopsy Carried Out.							No Autopsy Carried Out.						
		Stillbirths.			Neonatal Deaths.			Total.	Stillbirths.			Neonatal Deaths.			Total.
		Premature.	Mature.	Total.	Premature.	Mature.	Total.		Premature.	Mature.	Total.	Premature.	Mature.	Total.	
Asphyxia	1949	4	7	11	1	2	3		—	8	8	—	—	—	
	1950	8	41	49	6	3	9	72	10	15	25	3	—	3	36
Injury	1949	3	14	17	1	1	2	57	—	3	3	—	—	—	11
	1950	2	25	27	2	9	11		—	6	6	1	1	2	
Malformation	1949	1	4	5	—	1	1		1	3	4	—	—	—	
	1950	6	3	9	1	4	5	20	2	2	4	—	1	1	9
Premature	1949	1	—	1	1	—	1		—	—	—	4	—	4	
	1950	4	—	4	5	—	5	11	5	—	5	6	—	6	15
Toxæmia	1949	1	3	4	—	—	—		1	1	2	—	—	—	
	1950	2	3	5	—	—	—	9	5	6	11	1	—	1	14
Rh incompatibility ..	1949	1	3	4	—	—	—		—	2	2	—	—	—	
	1950	4	7	11	—	10	10	25	—	1	1	—	—	—	3
Others	1949	—	1	1	1	1	2		1	—	1	—	—	—	
	1950	2	3	5	1	10	11	19	—	2	2	1	—	1	4
Unknown	1949	2	8	10	—	—	—		2	14	16	1	1	2	
	1950	9	9	18	1	8	9	37	8	25	33	4	1	5	56
Total	1949	13	40	53	4	5	9		5	31	36	5	1	6	
	1950	37	91	128	16	44	60	250	30	57	87	16	3	19	148
Grand total ..	—	50	131	181	20	49	69	250	35	88	123	21	4	25	148

Note that of cases in which autopsy was performed, the cause of death remained unknown in only 14% (37 out of 250), whereas of cases in which autopsy was not performed, the cause was obscure in 37% (56 out of 148).

The series is a relatively small one, but it is believed that the information it reveals may be of interest to medical practitioners in general, and possibly of some assistance to others who may be contemplating a similar investigation on a more extensive scale in their own States.

Method of Inquiry.

Definitions.

The following definitions have been adopted.

"Stillbirth" is the birth of any child weighing two pounds twelve ounces or more which has issued from its mother but did not breathe or show any other signs of life.

"Premature birth" is the birth of any child weighing five pounds eight ounces or less at birth, but not less than two pounds twelve ounces.

"Pre-viable infant" is any infant weighing less than two pounds twelve ounces at birth.

"Neonatal death" is death occurring within the first four weeks after birth.

Data Available.

Within forty-eight hours of the occurrence of a stillbirth or premature live birth the medical practitioner who was in attendance is obliged by State law to submit a report on it to the Commissioner of Public Health on a special form distributed for this purpose.

Five millilitres of maternal blood are sent to the public health laboratory and subjected to the following investigations: (i) Wassermann test, (ii) blood grouping, (iii) Rh typing.

In certain proclaimed districts in the metropolitan area all stillborn infants are submitted to a post-mortem examination by a medical practitioner appointed by the Commissioner (the vast majority of these examinations have been conducted by the same practitioner, A.T.P.). At his discretion sections from one or more organs are sent to the public health laboratory for histopathological examination.

All these reports, clinical, post-mortem, histological and serological, are eventually submitted to the Department of Public Health, where they have been recorded and tabulated under the supervision of a medical officer (D.J.R.S.).

System of Classification.

A study of the first 100 reports received made it evident that the adoption of some broad basic classification of presumptive causes of death was necessary, in order to facilitate the recording of the facts available and to simplify the subsequent presentation of tabulated data. After perusal of the relevant literature and consideration of the report on neonatal mortality and morbidity released by the British Ministry of Health in 1949, the following provisional classification was devised: asphyxia, injury, malformation, prematurity, toxæmia of pregnancy, Rh incompatibility, others unknown.

Each of these categories is capable of further analysis with a view to revealing the frequency of individual factors which are likely to have contributed to the main cause.

Comments on Causes.

Asphyxia.

The term "asphyxia" when applied to the problem of fœtal and infant mortality implies a state of oxygen insufficiency existing before, during or after birth. It is used synonymously with the terms "hypoxia", "anoxia" or "anoxæmia", and is not necessarily attributable to respiratory deficiency or obstruction. During the prenatal period the fœtus is dependent on maternal oxygen, which may be diminished from anæmia or from cardiac or pulmonary disease. Further, at this time abnormalities of the placenta or umbilical cord may interfere with the transference of oxygen to the fœtus; or the fœtus itself, by reason of cardio-vascular malformation, may be unable to distribute available oxygen in an efficient manner. During labour, very prolonged uterine contraction, premature separation of the placenta and compression of the cord may reduce the supply of oxygen, while undue cranial stress may damage the nerve centres responsible for the subsequent initiation of respiration. Immediately after birth anoxia may result from the non-establishment of respiratory move-

ment, obstruction of the air passages from *liquor amnii* or meconium, or congenital anomalies of lungs, diaphragm *et cetera*.

Injury.

Injury to the fœtus occurs almost exclusively during the process of parturition, and its causation is essentially mechanical. Few, if any, fœtal structures are immune to birth trauma, but by far the commonest and most lethal group of injuries are those involving the cranium and its contents. Relative disproportion, malpresentation, excessive moulding of the head from prolonged labour, too rapid moulding in precipitate labour, application of forceps, breech extraction and other similar factors can cause distortion of the fœtal skull beyond the limits of its normal plasticity and result in rupture of the dural septa with dangerous intracranial hæmorrhage from torn venous sinuses. With less gross distortion the septa may remain intact, but kinking of major veins may set up sufficient stasis to result in bleeding or scattered areas of cerebral softening. Tentorial tears involving the lateral and straight sinuses are particularly dangerous, because of subtentorial hæmorrhage causing compression of the medulla. Tears of the *falx cerebri* also lead to serious results. Subarachnoid hæmorrhage is not usually fatal unless extensive. Hæmorrhage into one or other of the ventricles is commonest in prematurity and highly lethal, while scattered punctate or petechial hæmorrhages have a variable prognosis and are commonly associated with asphyxia.

Malformation.

The fœtus is liable to a wide range of malformations, but comparatively few of these seriously jeopardize its chances of a separate initial existence. Several others, however, although they may permit of live birth, are incompatible with continued existence long beyond the neonatal epoch. Anencephaly, *spina bifida*, hydrocephalus, cardio-vascular abnormalities, atresia of essential passages and congenital absence of various structures are a few general examples of conditions which comprise major hazards. The ætiology of malformations is still the subject of investigation and study, but in general, at the present time, they constitute the so-called "irreducible minimum" which is beyond the control of those striving to reduce the rate of stillbirth and infant death. As reduction in other controllable causes is effected, however, the influence of fœtal malformation will assume much greater importance.

Prematurity.

A definition of prematurity based on a weight criterion is less unsatisfactory than one based on the period of gestation. Amenorrhœa may be delayed, and the height of the uterus is variable; hence the more or less universal introduction of the two and three-quarter to five and a half pound rule. Though an improvement, even this definition has limitations. A five-pound baby may be quite mature and a two-and-a-half-pound baby is not necessarily "pre-viable". Nevertheless the new definition is more useful and informative than its predecessor.

The tissues of the premature infant are especially fragile. They are therefore more susceptible to the ill effects of oxygen deprivation and mechanical injury. Owing to immaturity of their vasomotor, respiratory and other centres, they are capable of but feeble response to adverse circumstances. Prematurity alone, on the grounds of physiological inadequacy, may be sufficient to prohibit survival. Cerebral damage and asphyxia are the main risks during premature births and account for most of the deaths in the early neonatal period, while after the first week of life these children commonly die because of infection. Furthermore, the circumstances under which premature births take place are peculiarly prejudicial to survival; for example, many are associated with toxæmia of pregnancy, ante-partum hæmorrhage, malformation, or multiple pregnancy. It is thus evident that the factors operating towards a fatal outcome among premature infants are usually complex, multiple and complementary.

Toxæmia of Pregnancy.

By the term "toxæmia of pregnancy" is meant the condition characterized by the familiar triad albuminuria, hypertension and œdema. For the purpose of the inquiry cases of albuminuria alone or of hypertension (systolic pressure, over 140 millimetres of mercury; diastolic pressure, over 90 millimetres) alone have been included but cases of accidental hæmorrhage alone have been excluded. Professor F. J. Browne, during a recent lecture on the subject in Perth, repeated that hypertension was the early sign and that when the systolic arterial pressure attained 180 millimetres of mercury renal arteriolar spasm resulted in kidney damage and albuminuria, while the spiral arteries in the placenta when similarly affected could cause retroplacental bleeding or "accidental hæmorrhage" and fœtal death. Disturbance of maternal fluid balance would obviously also involve the fœtus, while the fœtal risk associated with the disaster of eclamptic fits in the mother can readily be appreciated. Nevertheless, there is a considerable proportion of cases in which a mildly toxæmic mother delivers a full-term stillborn child despite a normal labour and an apparently unaffected placenta. The nature of the lethal factor here is unknown.

Rhesus Incompatibility.

The term "rhesus incompatibility" includes *hydrops fœtalis*, *icterus gravis neonatorum* and hæmolytic anæmia of the newborn. They are merely grades or degrees of the same disorder, which is characterized essentially by hæmolysis of rhesus-positive fœtal erythrocytes by antibodies developed in rhesus-negative maternal blood. The effects in the fœtus include anæmia, hyperbilirubinæmia, extramedullary erythropoiesis (erythroblastosis), œdema, and in some cases neuronal degeneration and yellow staining in the basal ganglia ("kernicterus"). *Hydrops fœtalis*, which is characterized by general œdema with effusions, hepatosplenomegaly and other changes, is fatal before or soon after birth. *Icterus gravis* is a less consistent cause of stillbirth, but often causes death in the neonatal period, while the mortality from congenital hæmolytic anæmia is relatively small. While rhesus incompatibility is known to be responsible for many intrauterine deaths, stillbirths and premature births, the precise frequency of the association remains to be determined, particularly in specific races and localities.

Preliminary Findings.

A total of 457 consecutive notifications of stillbirths, neonatal deaths and premature births has been recorded at the Department of Public Health over a period of seventeen months, that is, from August 1, 1949 (when autopsies in Perth became compulsory), to December 31, 1950.

Of this number 29 have been omitted from the analysis, either because the data provided were inadequate or because the fœtus was under two pounds twelve ounces in weight and therefore "pre-viable".

Of the remaining 428 cases, 250 relate to infants brought to autopsy.

The series of 428 comprises 304 stillbirths and 94 neonatal deaths (including premature births), and 30 premature births in infants who survived.

It is emphasized that while stillbirths and premature births are notifiable by State law and subject to special inquiry, neonatal deaths are not. Figures for the latter must therefore be incomplete.

Stillbirths.

Of the 304 stillbirths (which included 181 involving autopsies) the probable causes were as follows: asphyxia, 93 (30·5%); injury, 53 (17·4%); malformation, 22 (7·2%); prematurity, 10 (3·2%); toxæmia, 22 (7·2%); rhesus incompatibility, 18 (5·9%); other miscellaneous causes, 9 (2·9%); undetermined, 77 (25·3%).

Neonatal Deaths.

Of the 94 neonatal deaths (which included 69 involving autopsies) the probable causes were as follows: asphyxia, 15 (15·9%); injury, 15 (15·0%); malformation, 7 (7·4%); prematurity, 16 (17·0%); toxæmia, 1 (1·0%); rhesus incompatibility, 10 (10·6%); infection, 9 (9·5%); other miscellaneous causes, 5 (5·3%); undetermined, 16 (17·0%).

Premature Births.

The factors associated with prematurity in the 30 premature infants who survived were as follows: multiple pregnancy, 8; maternal toxæmia, 5; *placenta prævia*, 2; rhesus incompatibility, 1; unknown or uncertain, 14.

Obstetrical Difficulties.

The various obstetrical difficulties recorded were as follows: malpresentation, 92; multiple birth, 27; cord complications, 36; ante-partum hæmorrhage, 52; maternal toxæmia, 60; rhesus-negative mothers, 53.

Types of Injury.

The following specific cranial and intracranial injuries sufficient to cause death were revealed at autopsy: tear of *tentorium cerebelli*, 25; tear of *falx cerebri*, 5; subdural hæmorrhage, 5; subarachnoid hæmorrhage, 5; intraventricular hæmorrhage, 6; rupture of great cerebral vein, 2; depressed fracture of parietal bone, 2; "intracranial hæmorrhage" (unspecified), 6; craniotomy, 1; total, 57.

Prematurity.

Of the 398 stillbirths and neonatal deaths, 129 (or 32·4%) were in premature infants. The apparent causes of prematurity were as follows: ante-partum hæmorrhage, 22; toxæmia of pregnancy, 12; ante-partum hæmorrhage and toxæmia of pregnancy, 5; multiple pregnancy, 12; rhesus incompatibility, 9; fœtal malformation, 13; maternal disease other than toxæmia, 1; unknown, 55 (42·6%).

Of the 129, 82 were stillbirths (including 17 examples of prenatal death) and 47 were neonatal deaths.

The respective birth weights were as follows: under three pounds, 19; three to four pounds, 44; four to five pounds, 31; five to five and a half pounds, 17; not stated, 18.

Blood Examination.

The results of examination of maternal blood in those cases in which specimens were provided are as follows. (i) Rhesus typing: Rh-negative, 53 (25·5%); Rh-positive, 155 (74·5%). (ii) Blood grouping: AB, 1 (0·8%); A, 52 (45·2%); B, 9 (7·8%); O, 53 (46·1%). (iii) Wassermann test: negative result, 179 (96·7%); positive result, 6 (3·3%).

Conclusion.

1. The preliminary findings following an investigation into the causes of stillbirth in Western Australia appear to conform to the following already well-known pattern: (i) A group associated with, or attributable to, prenatal factors, such as toxæmia of pregnancy, breech presentations, ante-partum hæmorrhage and rhesus incompatibility. (ii) A group associated with special obstetrical difficulties, such as occipito-posterior positions, breech presentation, multiple pregnancy and ante-partum hæmorrhage. (iii) A relatively large undetermined group of causes resulting in prenatal death, premature birth and unexplained stillbirth.
2. Post-mortem examination has proved of value in revealing the cause of stillbirth and neonatal death in Western Australia.

Acknowledgement.

We are indebted to Dr. Linley Henzell, Commissioner of Public Health in Western Australia, for permission to publish this preliminary report and his encouragement in its preparation; to all the members of the Infant Mortality Committee of the State Health Council, and in particular to Dr. Geoffrey Thompson for his invaluable advice; and to all the members of the medical profession in Western Australia who have provided the data upon which this report is based.

THE NEUROGENIC FACTOR IN RHEUMATIC INFLAMMATION.[1]

By MICHAEL KELLY,
Melbourne.

WHAT is rheumatism? Can we as scientists accept such a term, which has been described as a grab-bag containing a hundred unrelated and ill-understood disorders? The layman uses it with a confidence born of ignorance, in the broad anatomical sense of pain in muscles and joints. We, possessed of a little learning, can neither adopt the term with confidence nor reject it out of hand. As students we learned to think of its grosser manifestations—rheumatic fever, rheumatoid arthritis, osteoarthritis—as unrelated constitutional diseases characterized by local areas of inflammation in fibrous and synovial tissues. After graduation we have to learn for ourselves about the myriad minor manifestations of rheumatism—acute, subacute or chronic inflammation of fascia, ligaments, tendons, bursæ, synovial sheaths, and articular tissues—which comprise the bulk of the cases encountered in daily practice.

Common Features.

These disorders have many features in common; they often follow infection or injury, but more often they seem to arise spontaneously. They cannot be clearly separated from the polyarthritides; they present every gradation from a single fibrositic spot to multiple articular and fibrous lesions. In each individual lesion the same pathological process is at work, whether the disorder is widespread or localized, and I am going to call this process rheumatic inflammation. A notable feature of rheumatic inflammation is its tendency to spread. Sometimes the spread is by direct extension and sometimes by the appearance of new lesions at a distance. This has never been satisfactorily explained, nor has the rapid disappearance of many lesions.

By the observation of individual lesions a good deal can be learned about rheumatic inflammation. The more cases of rheumatism I have encountered, the more clearly have I discerned the features they have in common. Here I shall mention a few of these features and put forward a hypothesis to explain them.

Rheumatic Tenderness.

There are three phases in rheumatic inflammation: (i) tenderness without visible change, (ii) cellular hyperplasia and secretion of fluid, (iii) fibrosis. The well-known changes in the second and third phases can be discerned with the naked eye or the microscope, but the first stage can be recognized by clinical examination only. As a rule the tenderness comes and goes within a few hours, and no swelling or other sign of inflammation is seen. At this early stage the tender tissues show no microscopic change. But tenderness is a positive sign that means a real change —the beginning of rheumatic inflammation. If we can discover, therefore, the mechanism that induces the tenderness, we shall be close to comprehending the pathogenesis of rheumatism.

Tenderness and hyperæmia are the earliest manifestations of inflammation no matter what its cause; they signify nothing but damage to the tissues. In rheumatism no agent has yet been identified that can directly damage the tissues, and none of the theories that attempt to explain the inflammation are completely satisfying. We are apt to forget that the tissues may be damaged indirectly as well as directly, and some evidence suggests that the indirect or neurogenic factor is important in rheumatic inflammation.

Hyperæmia.

Adami (1907), Lewis (1927) and others have pointed out that there are two elements in inflammatory hyperæmia and exudation: (i) direct damage to vessel walls, (ii) reflex nervous vasodilatation.

[1] Read at the annual meeting of the Australian Association of Clinical Pathologists, Brisbane, May 27, 1950.

Inflammation induces vasodilatation in nearby arterioles through axon reflexes (Figure I). The impulses travel by the finest branches of the somatic sensory nerves and not

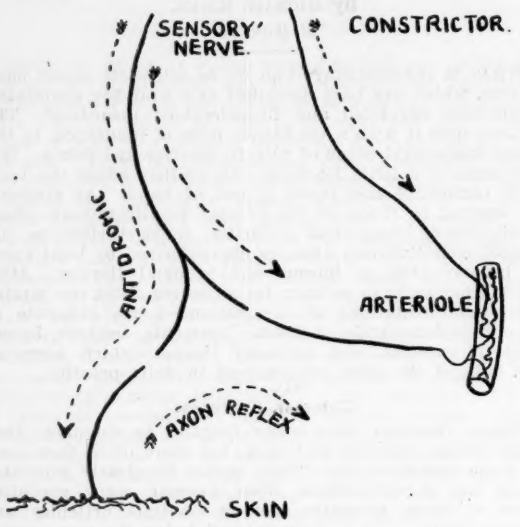

FIGURE I.

Antidromic impulses and axon reflex. Nerves which mediate hyperæmia are separate from sympathetics. (Lewis, 1927.)

by the sympathetic (Ninian Bruce, 1912; Langley, 1923; Lewis, 1927). Bayliss (1900) demonstrated antidromic vasodilatation in the deep tissues in addition to the skin (Figure II).

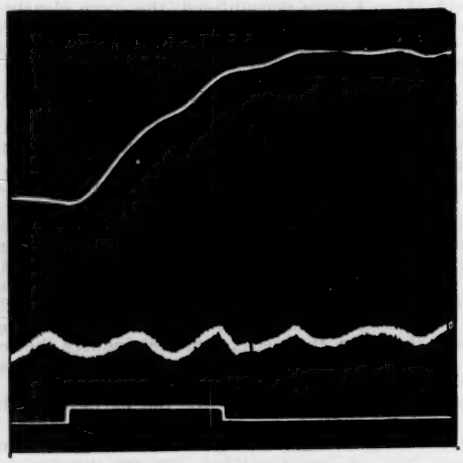

FIGURE II.

Vasodilators in posterior nerve roots. Upper record, limb volume; lower record, blood pressure. Signal: stimulation of the peripheral end of a lumbar posterior nerve root eight days after section between the spinal cord and the ganglion. Note dilatation of limb (indicative of vasodilatation) without change in the level of the blood pressure. (Bayliss, 1900.)

Hyperalgesia.

Lewis (1936) has shown also that hyperalgesia, like hyperæmia, may be generated in two ways—(i) by direct damage to the tissues, (ii) by wider liberation of a "pain substance" by axon reflexes through sensory nerves.

Hyperalgesia appears in the field of a sensory nerve after antidromic stimulation; when the ulnar nerve has been stimulated at the elbow the ulnar field in the hand becomes hyperalgesic; and after faradization of one of its digital branches the same cutaneous area becomes hyperalgesic (Figure III).

FIGURE III.

Antidromic and reflex hyperalgesia. Faradization of digital nerve at *A* gives hyperalgesia of area indicated by broken line. Faradization of ulnar nerve at *C* gives hyperalgesia of same area. Faradization of digital branch of radial nerve gives hyperalgesia of area indicated by continuous line. (Lewis, 1936.)

Though antidromic vasodilatation has been known for eighty years, physiologists have doubted whether it plays any part in normal tissue reactions. But *herpes zoster* is well known as an example of neurogenic inflammation in the skin. Inflammation, Adami stated, may induce vasodilatation in distant regions that are supplied by the same segment; wealing and blistering of the skin can be induced by hypnosis.

Deep Tenderness.

Tenderness confined to the deep tissues may not be due to local damage, but it may be referred from deep somatic lesions or from viscera. When Kellgren (1939) injected an irritant into the dorsal muscles of the forearm, pain was felt for a few minutes in the hand, and for the duration of the pain the deep tissues of the back of the hand became tender. Similarly an injection into the brachialis muscle was followed by evanescent tenderness of the flexor mass of muscles. Now what does this tenderness signify?

The orthodox view—as expressed by Cohen (1947) and others—holds that normal afferent sensory impulses from the tender area have been intensified by summation with impulses from the artificial lesion, to give a result which the brain misinterprets as tenderness. But Sinclair, Weddell and Feindel (1948) suggest that, when tenderness appears in visceral disease, impulses from the viscus have liberated a pain substance by antidromic reflex action (Figure IV). The branching sensory nerves come from posterior root ganglia; the afferent impulses may turn at the axon branchings, or they may be relayed in the cord. I believe that this interpretation is true for somatic lesions also; instead of the viscus (E) or the skin (C) we might read another muscle or a joint or any other deep tissue (Figure V). Thus deep tenderness tends to generate further deep tenderness and it is patchy.

Now, the tenderness of the earliest stage of rheumatic inflammation closely resembles the tenderness that is referred from a somatic lesion. We know well how rheumatic tenderness appears in patches separated by long gaps of normal tissue, and how some of the tender areas return quickly to normal while others persist and proceed to swelling. When spread is rapid there may be no semblance of order, but in less acute cases (and more especially when pain spreads after a localized injury) an orderly method of spread can be distinguished.

Local Analgesia.

Local analgesia has proved especially suitable for investigation for two reasons: (i) widespread pain and tenderness

may disappear on anæsthetization of a small area; (ii) in many cases the relief is lasting.

With the aid of local analgesia, rheumatic tenderness may be differentiated into the following two groups: (i) sharply localized primary lesions; (ii) reflex or diffuse tenderness that disappears when the primary lesion is anæsthetized. It often surrounds the primary lesion, but may be in patches at a distance from it.

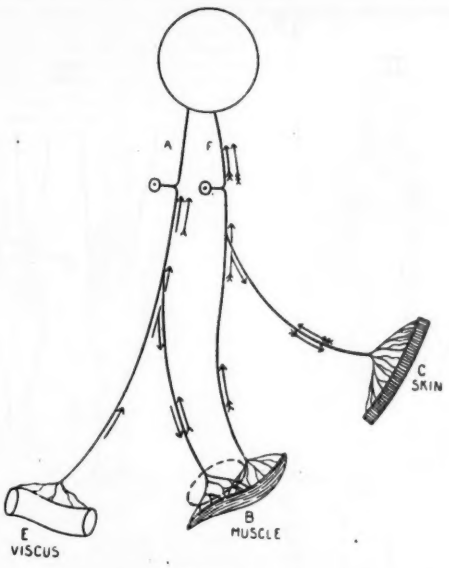

FIGURE IV.

Reference in stages. Impulses from viscus set up a chemical disturbance in muscle or in skin, either via axon branchings or through relays in cord. Impulses from the disturbed muscle then pass up the second branched axon to the skin. (Sinclair, Weddell and Feindel, 1948.)

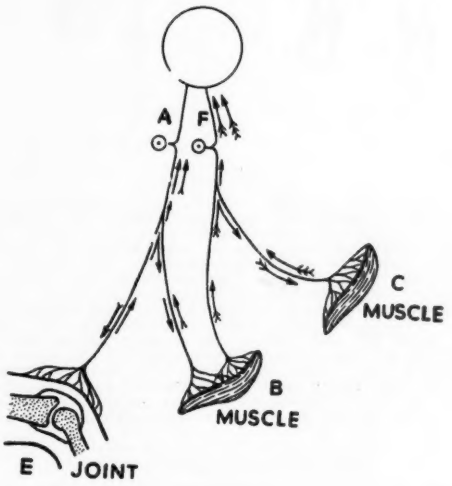

FIGURE V.

Adapted from Figure IV. Reference in stages from joint to muscle, or from muscle to joint.

Primary lesions may occur anywhere in muscle or fibrous tissue, but they favour certain regions and they prefer musculo-tendinous junctions. About 65% of them are found at the common sites shown in Figure VI. Each lesion has a fairly constant zone of reference; this is why the patterns of rheumatic pain are fairly constant from case to case.

Pain in Forearm and Hand.

Infiltration of a lesion in the common site in the extensor muscles of the forearm (Figure VI, 3) may abolish pain and tenderness in the hand or the wrist. In Case I (Figure VII) three areas of tenderness disappeared, one surrounding the lesion, but the others a good distance proximal and distal to it. In a series of cases previously reported (Kelly, 1944*b*) all the primary lesions were near the elbow joint,

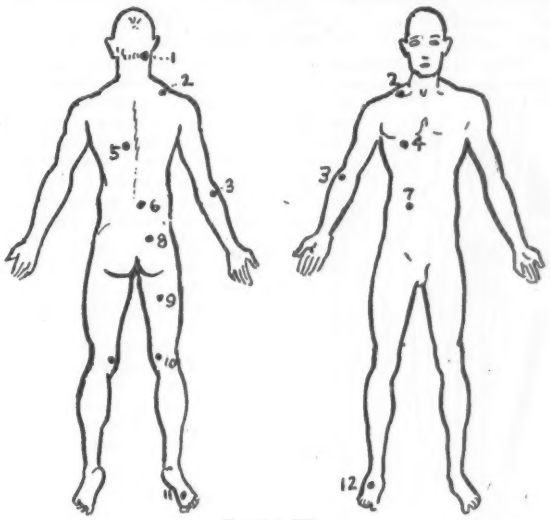

FIGURE VI.

Common sites for myalgic lesions, numbered as in Table I.

TABLE I.

Radiation of Pain from Common Sites. (See Figure VI.)

Number.	Site.	Type of Pain.
1	Angle between trapezius and sterno-mastoid.	Stiff neck; headache; facial neuralgia.
2	Trapezius or supraspinatus at mid-scapular line.	Pain on abduction of humerus brachial neuralgia.
3	Extensors below lateral epicondyle.	Pain in forearm and hand.
4	Anterior end of intercostal space.	Pleurodynia; intercostal neuralgia.
5	Posterior end of intercostal space.	Pain in back of chest, often radiating to front; pleurodynia, intercostal neuralgia.
6	Lumbar sacrospinalis.	High lumbar backache.
7	Edge of *rectus abdominis*.	Abdominal pain.
8	*Gluteus maximus* in sacro-iliac region.	Lumbo-sacro-iliac pain; may radiate to iliac fossa or down to gluteal fold.
9	Upper third of hamstrings.	Sciatic pain.
10	Adductor tubercle region.	Pain in knee.
11	*Flexor brevis hallucis*.	Pain along inner side of foot and big toe.
12	*Extensor brevis digitorum*.	Pain in instep.

but their effects radiated in various ways. This experience has now been amply confirmed in nearly 200 cases. In another case the pain and tenderness of an intractable tenosynovitis of the thumb extensors vanished promptly on infiltration of a lesion in *extensor communis digitorum*. The pain did not recur and the swelling and crepitus subsided promptly.

Pain in Shoulder.

Infiltration of a lesion at a common site in the trapezius (Figure VI, 2) may abolish tenderness of the subacromial bursa and the deltoid muscle. A series of cases reported

some years ago (Kelly, 1942) showed individual variations but fairly constant patterns of pain in the deltoid. More than once a chronic effusion into the subacromial bursa has subsided within a few days of treatment of the lesion in the trapezius. Shoulder pain may extend to the neck by the development of a lesion in the nuchal region (Figure VI, 1) or to the forearm by the development of lesions in the extensor muscles. In other cases pain in the forearm extends to the shoulder.

Pain in Knee.

Pain and tenderness in joints are frequently referred from muscular lesions. The commonest cause of pain in the knee is a lesion (Figure VI, 10) just above the medial femoral condyle (Kelly, 1944*a*). Referred pain and tenderness of the joint may vanish immediately on injection of procaine. In several cases an intractable effusion has disappeared within two days.

FIGURE VII.

Case I. Lesion at *A* was surrounded by diffusely tender area (hatching). Another area of tenderness was defined above the lateral condyle, and a third on the palmar aspect of the wrist. On anæsthetization of *A*, tenderness of all three areas vanished.

This rheumatic tenderness, therefore, is produced by a reflex, and when it persists and proceeds to swelling the reflex mechanism is still largely responsible. In joints and other mobile regions movement intensifies both the hyperalgesia and the secretion of fluid. But in many cases tenderness of a swollen joint, bursa or synovial sheath has vanished when the primary lesion has been infiltrated.

Case II (Figure VIII) was one of polyarthritis following rubella; widespread tenderness above and below the knee vanished when the lesion in the common site was treated. The swollen knee joint rapidly returned to normal, though all the other joints remained painful and swollen.

In Case III (Figure VIII) the pain and tenderness of an acutely distended knee joint vanished when a tender spot posterior to the joint was anæsthetized; at the same time severe cramps in an acutely swollen foot were abolished by injecting a tender spot in *extensor brevis digitorum*.

In Case IV (Figure VIII) a small sore on the foot that soon healed was followed by painful distension of the ankle joint for two weeks, which prevented the boy from putting his foot to the ground and kept him awake every night with severe cramps in the calf. Immediately after injection of a tender spot in the calf he walked without pain, and next day the ankle was no longer swollen; there was no recurrence.

In Case V (Figure VIII) recurrent pain in the foot of two years' duration was associated with distension of the talo-navicular joint and great tenderness of the tuberosity of the navicular. Immediately after injection of a tender spot in *extensor brevis digitorum*, the tenderness vanished and the boy walked without pain. Next day the swelling had gone, and there has been no recurrence in three years.

Spread of Pain.

When somatic pain and tenderness persist after injury, a lesion of this kind may be responsible. Spread of pain, whatever its original cause, is facilitated by the development of new lesions, usually at common sites. Thus a contusion of a muscle (Figure IX) may be followed by persistent pain and tenderness which prove to be due to a primary lesion near the musculo-tendinous junction. The tenderness may include part of another muscle, in which a second lesion develops, and its area of radiation may spread in patches down the limb, with perhaps the development of a third lesion.

We have seen that pain in the forearm may extend to the shoulder, and pain in the shoulder may extend to the forearm and hand. Raney and Raney (1949) recently described a series of cases in which tender spots were present in the neck, the shoulder girdle and the forearm (Figure X). They attributed them to pressure on sensory roots and therefore recognized their neural origin. Steinbrocker, Spitzer and Friedman (1948) have described a reflex mechanism that connects periarthritis of the shoulder with painful swelling of the wrist or hand. I believe that such a mechanism facilitates the spread of rheumatic inflammation everywhere.

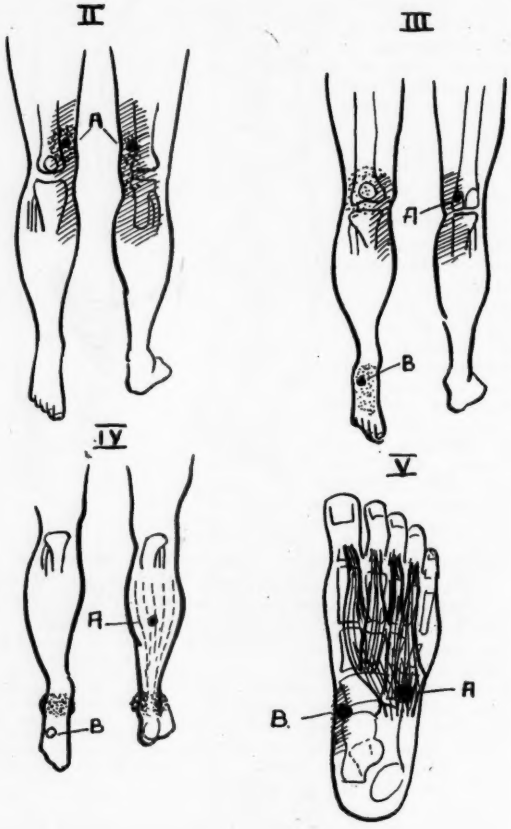

FIGURE VIII.

Cases II, III, IV and V. II. Polyarthritis following rubella: *A*, lesion in common site (right knee); stippled area, swelling on medial aspect of knee; hatched area, deep tenderness extending to thigh and lower part of leg on posterior and medial aspects. III. Arthritis of knee and foot: *A*, lesion near medial condyle of femur; stippled area, swelling of knee; hatched area, diffuse tenderness; *B*, lesion on dorsum of acutely swollen foot. IV. Effusion into ankle joint with painful lesion in calf: *A*, lesion in calf muscle; *B*, recently healed sore on dorsum of foot; stippled area, swelling of ankle. V. Effusion into talo-navicular joint with lesion in *extensor brevis digitorum*: A, lesion in muscle; *B*, acute tenderness at tip of navicular; hatched area, diffuse tenderness on medial aspect of talo-navicular joint.

Bilateral Symmetry.

Exact symmetry of rheumatic spread often gives striking support to the neurogenic theory. This evidence cannot be denied; Sir James Paget (1879) stated years ago that symmetry of disease must depend on nervous connexions rather than on any assumed identity of chemical composition. When pain persists after a simple injury it sometimes spreads to the opposite side.

After an experimental injection into the medial head of my right gastrocnemius muscle, pain and deep tenderness

extended all over the calf and lower third of the thigh. Twelve hours later a pale reflection of the pain was felt in exactly symmetrical areas of the left thigh and calf, and the painful areas were tender to pressure.

Case VI illustrates the symmetry of the rheumatic process and its progress by regular jumps to involve other joints.

In a case of rheumatoid arthritis of thirteen years' duration, the right knee was distended with fluid, but the left knee and the ankles had not been involved. The fluid was aspirated from the right knee with a trocar. Two days later the left knee became very painful, with distension of a popliteal bursa and widespread tenderness posteriorly (Figure XI). The tenderness disappeared for a few hours after anæsthetization of an extremely tender spot in the gastrocnemius muscle. Next day a large part of the calf was acutely tender, but spontaneous pain was felt chiefly in the ankle. For two days the painful ankle presented a normal appearance; then it suddenly became distended with fluid.

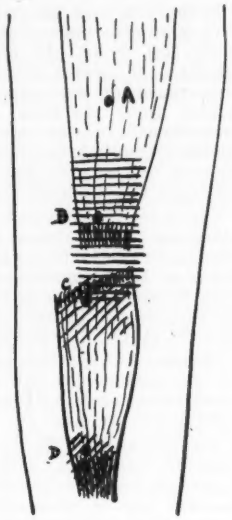

FIGURE IX.

Spread of tenderness from injury: *A*, contusion of muscle; *B*, myalgic lesion at musculo-tendinous junction; horizontal hatching, diffuse tenderness associated with *B*, which spreads to include part of second muscle; *C*, second myalgic lesion; oblique hatching, two areas of diffuse tenderness referred from *C*; *D*, third myalgic lesion.

Discussion.

These scattered observations do but little justice to the large volume of evidence which implicates the nervous system in the persistence and spread of rheumatic inflammation. By implication they suggest that rheumatic inflammation is a non-specific change in the tissues which resembles the changes due to other kinds of damage.

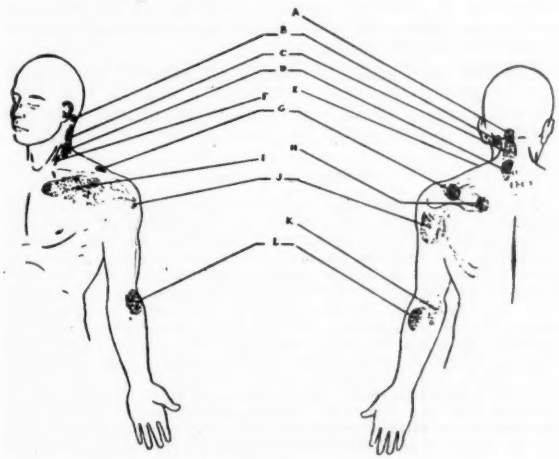

FIGURE X.

Tender spots in neck, shoulder girdle and forearm (Raney and Raney, 1948): *B*, *G* and *L* correspond with sites 1, 2 and 3 (Figure VI).

The neurogenic factor is not proposed as a complete explanation of rheumatism; it is suggested rather as a means through which a combination of causative factors may act to damage the tissues. The theory is not contradicted by the discovery of the importance of hormonal factors, for there are other instances in plenty of hormonal disturbances which act on tissues through their nerve-endings (Wyburn-Mason, 1950).

We should not forget that a neurogenic origin for rheumatism was widely accepted by the leaders of medical thought from 1750 until the end of last century (Cullen, 1810; J. K. Mitchell, 1831; Laycock, 1864; Weir Mitchell, 1872; Liveing, 1873; Charcot, 1877; Paget, 1879; Ord, 1880; Duckworth, 1884; Latham, 1886; Spender and Garrod, 1897; *et cetera*). Déjerine (1926) never abandoned the neural concept of arthritis; he has been supported by Leriche (1928), Lichtwitz (1944), Martin (1944), Morrison and others (1948), Steinbrocker, Spitzer and Friedman (1948), and many others.

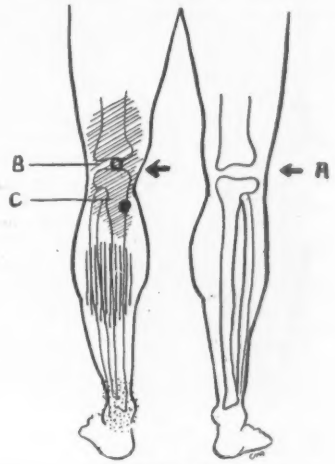

FIGURE XI.

Case VI. Rheumatoid arthritis: spread of exacerbation after injury to knee. *A*, site of puncture of right knee; *B*, cystic swelling behind left knee, surrounded by diffuse tenderness (oblique hatching); *C*, myalgic lesion in gastrocnemius; vertical hatching, diffuse tenderness of calf after infiltration of *C*; stippling, pain (and later swelling) in ankle which radiated from calf.

Summary.

1. Hyperalgesia, hyperæmia and exudation of fluid may be mediated through reflex action; nervous impulses therefore play an important part in the spread of inflammation.

2. Referred pain from viscera or from somatic tissues may be accompanied by tenderness that is due to the reflex liberation of a "pain-substance".

3. In all the rheumatic disorders there are many common factors. Tenderness is the earliest stage; exudation of fluid and cellular hyperplasia are further stages of the same process.

4. With local analgesia the tenderness can be resolved into two elements: (i) localized primary lesions; (ii) diffuse reflex tenderness which disappears when the primary lesion is anæsthetized. Patches of reflex tenderness may be at a distance from the primary lesion, separated from it by normal tissues.

5. In the zone of reflex tenderness (which frequently includes joints) exudation of fluid may occur. Thus effusion into a joint may be a reflex effect.

6. Any area of rheumatic tenderness may act as a focus for further reflex spread. In many cases an orderly spread can be observed.

7. Therefore, in the spread of rheumatic inflammation, neurogenic factors play a predominant part.

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DIFFUSE SCLERODERMA, WITH REPORT OF A CASE SHOWING MULTIPLE LESIONS.

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Melbourne.

SCLERODERMA is the term applied to a peculiar induration of the skin, which may be the cutaneous manifestation of a widespread disease of collagenous tissue, but which sometimes represents merely the secondary effect of a variety of other pathological processes.

For the latter conditions Parkes Weber (1946) has suggested the term symptomatic scleroderma, grouping under this heading the changes which may occur in association with such diseases as chronic eczema, urticaria, idiopathic amyloidosis and thyreotoxicosis.

"Primary" scleroderma is found in circumscribed and diffuse forms, the former being exemplified in the ivory plaque or band of morphœa. Barber (1943) has reported its rare appearance on the buccal mucosa.

"Diffuse" scleroderma is a disease of unknown ætiology, in which skin changes may be associated with vasomotor disturbances and lesions in the viscera. Terms such as "diffuse collagen disease" (Klemperer *et alii*, 1942) and "progressive systemic sclerosis" (Goetz, 1945) have been used to emphasize the widespread nature of this disease.

Diffuse scleroderma may occur at any age, but the incidence is greater in the fourth and fifth decades, and it is more common among females than males.

In adults the disease generally presents with tightness of the skin, which limits movement and facial expression. The subcutaneous fat disappears and the skin is stretched over bony prominences, becoming firmly adherent to underlying soft parts. In other cases Raynaud's phenomena appear in the hands, which later develop the stiff, claw-like appearance of sclerodactylia. Allen Barker and Hines (1946) apply the term "acroscleroderma with Raynaud's phenomena" to this variant, and point out, by way of contrast, that in true Raynaud's disease the sclerodermatous changes appear at a late stage and involve only the parts in which the Raynaud's phenomena occur.

Occasionally symptoms arising from the visceral complications of this disease bring the patient to the physician. Ehrmann (1903) first described a narrowing in the lower third of the œsophagus due to scleroderma. Since that time it has been reported by others, including Rake (1931), Lindsay *et alii* (1943), Goetz (1945), Pugh *et alii* (1945), Bourne (1947) and Schwartz and Skinsnes (1949).

Weiss *et alii* (1943) reported nine cases of congestive cardiac failure due to "scleroderma heart disease", while more recent cases have been noted by Mathisen and Palmer (1947) and by East and Oram (1947).

Murphy *et alii* (1941) recognized the radiological appearance of pulmonary infiltration due to scleroderma, the diagnosis being subsequently confirmed at autopsy. Pulmonary lesions were present in three of Weiss's (1943) cases and in that reported by Mathisen and Palmer (1947).

The association of scleroderma with interstitial calcinosis is well known; this is sometimes called the Thibierge-Weissenbach syndrome.

Renal complications of diffuse scleroderma are uncommon, although albuminuria is often present at some stage of the disease. Bilateral focal cortical necrosis associated with scleroderma has been reported by Talbott *et alii* (1939).

Occasionally basement membrane thickening of glomerular capillaries resembles the "wire loop" appearance found in cases of disseminated *lupus erythematosus*. The relationship of scleroderma to *lupus erythematosus* has been debated by Klemperer *et alii* (1942), by Goetz (1945), and by Baehr and Pollack (1947), and in general there is some reluctance to assume a common ætiology on the basis of similar morphological appearances and occasional coincidence in the same patient.

The case here reported is unusual in some of the clinical manifestations of diffuse scleroderma and in the renal cortical necrosis, which together with cardiac failure caused death. For this reason it was thought worth reporting.

Report of a Case.

J.B., a male patient, aged forty-six years, was first examined in March, 1947, when he complained of weakness in the arms and breathlessness on exertion. He had received treatment for malaria in 1945, when his medical attendant noticed that the patient's hands appeared blue and swollen. The patient noticed no disability at this time, but six months later he found his hands becoming blue, stiff and painful, especially on cold days. During the subsequent winter he suffered episodes of pain and stiffness of the hands and fingers, which became white at these times. At the finger tips small, yellow, painful areas developed. In 1946 periarterial sympathectomy was performed upon both brachial arteries for the relief of "Raynaud's disease". After the operation his fingers remained stiff and blue, but the sores at the finger tips healed. He was able to do light work until six months prior to his admission to hospital, when he noticed his legs becoming weak and his knees and ankles stiffening. Over the last two months, he became "stiff all over" on resting and noticed breathlessness on exertion. His past history disclosed nothing of significance except that during his army service he had manipulated pneumatic drills daily for two periods of six and four months, in 1941 and 1942 respectively. His family history was uninformative.

On examination of the patient, the fingers of both hands were held in the position of rest. The skin over the digits

was smooth and tightly stretched so that it could not be wrinkled on pressure. It had a waxy, pale, slightly cyanotic appearance, while at the tips of the index, middle and ring fingers of both hands, small depressed scars were visible. The skin over the wrists and arms appeared to be of normal texture. The fingers could not be flexed into the palms, the most limited joints being the distal interphalangeal joints. Flexion and extension of the wrist, ankle and knee joints were also restricted to some degree. Movements of the wrists and fingers were accompanied by crepitus in the extensor tendon sheaths of both sides. This tenosynovitis was painless, and was found to be present to a greater or lesser degree at all subsequent examinations. The skin of the face, abdomen, thighs and legs appeared to be lacking in subcutaneous fat and was difficult to wrinkle over underlying structures, while scattered over the trunk and thighs were areas of vitiligo. The skin generally had a slight brown pigmentation, and occasional small spider angiomata were seen on the left palm and on the face.

No cardiac abnormality was found apart from occasional extrasystoles. The blood pressure was 112 millimetres of mercury, systolic, and 68 millimetres, diastolic. Absent pulsation in the right *dorsalis pedis* and posterior tibial arteries was noted, together with blanching of the feet on elevation and a slow return of colour on lowering, which was more pronounced on the right side. Examination of the lungs revealed a left pleural effusion, but no abnormality of the right lung was detected. The examination of the abdomen and central nervous system revealed no abnormality.

A diagnosis of diffuse scleroderma with Raynaud's phenomena was made. A biopsy of skin from the abdomen and pectoral regions disclosed the characteristic flattening of the rete pegs with a diffuse increase in collagen fibres and the absence of subcutaneous fat (Figure I).

At this stage of the disease no definite evidence of visceral lesions could be discovered, although the left pleural effusion raised the possibility of pulmonary involvement.

A barium meal examination revealed no lesion in the œsophagus or in the gastro-intestinal tract. The electrocardiogram was of normal configuration though low voltage.

An X-ray film of the chest taken before the onset of the illness in 1945 was available, and showed no lesion in either lung field. When the examination was repeated, the presence of the left pleural effusion was confirmed, and the right lung appeared normal.

Examination of the pleural fluid revealed it to be straw-coloured fluid, which on microscopic examination was found to contain numerous red cells and a few pus cells. No organisms were found in the fluid obtained from several aspirations, all cultures, guinea-pig inoculation and culture of fasting gastric contents giving negative results for tubercle bacilli.

Bronchoscopic examination failed to reveal any intrabronchial disease. At thoracoscopy, a pleural surface was seen on which white, thickened areas were interspersed with others of extreme vascular engorgement.

Other investigations gave the following results. No albuminuria was present, and no cells or casts were found in the urine. Blood examination revealed that the hæmoglobin value was 11·5 grammes *per centum*, and the leucocytes numbered 14,400 per cubic millimetre (62% neutrophile cells, 29% lymphocytes, 4% monocytes, 4% eosinophile cells and 1% basophile cells). The erythrocyte sedimentation rate was 32 millimetres in the first hour and 67 millimetres at the end of two hours. The Mantoux test produced a positive reaction. The Wassermann test produced no reaction, and cold agglutination was not detected. The plasma protein content was 6·7 milligrammes *per centum* (albumin 4·1, globulin 2·6 milligrammes *per centum*). The serum calcium content was 9·0 milligrammes *per centum* and the phosphorus content was 5·0 milligrammes *per centum*. X-ray examination revealed no bony changes.

The pleural effusion was aspirated on several occasions, and resolution occurred slowly over the succeeding four months. Subsequent radiological examinations revealed no evidence of disease in either lung field.

The temperature chart showed a daily elevation of 99° F. to 100° F. over the first six months, which was uninfluenced by penicillin administration. Thereafter occasional elevations occurred until the patient's discharge from hospital in November, 1947. At this time the disease appeared to have reached a static phase. Testosterone (10 milligrammes) was given daily by intramuscular injection for two months, with negligible effect.

The patient was able to work as a lift attendant, suffering greater disability during cold weather, until February, 1950, when further deterioration set in and he was again admitted to hospital. The skin everywhere had become tighter, restricting movement of all his joints, but particularly that of the hands, wrists and elbows. The fingers were now fixed in flexion and the palmar fascia in both palms was thickened and contracted, the appearance resembling Dupuytren's contracture. Tenosynovitis was present in the forearms and in both suprapatellar bursæ. Œdema of the ankles appeared and then rapidly became generalized, being

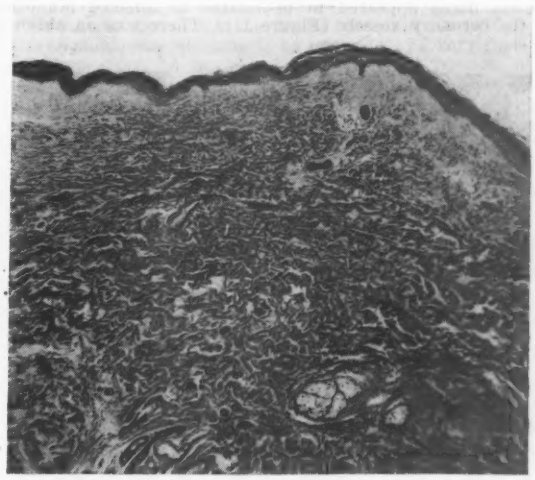

FIGURE I.

Low-power photomicrograph of skin biopsy from pectoral region, showing flattening of epidermis and collagenous hypertrophy throughout the dermis. (× 40.)

associated with ascites and a right pleural effusion. The heart at this stage rapidly increased in size, a third heart sound was audible at the apex, and later a loud pericardial friction rub developed over the precordium. The electrocardiogram revealed low voltage and flattening of the *T* waves with inversion of the *T* wave in Lead I. The blood pressure was 130 millimetres of mercury, systolic, and 95 millimetres, diastolic.

The empirical administration of DOCA, five milligrammes by intramuscular injection, together with ascorbic acid, one gramme intravenously each day for a week, was tried without appreciable benefit. The administration of para-aminobenzoic acid, two grammes every three hours, was commenced on the strength of a report of its success in the hands of Zarafonetis *et alii* (1950), but the patient, then *in extremis*, failed to respond to treatment.

During the last week of life the skin presented a livid hue interspersed with areas of vitiligo, and numerous spider angiomata were present over the face, neck and arms. The skin was thickened and pitted on pressure from the feet to the top of the head. The urinary output was reduced to only a few ounces during the week prior to death, and complete anuria prevailed during the last four days. Albumin was present in the urine, but there were no cells or casts. The blood urea level rose to 528 milligrammes *per centum*, and the patient died in cardio-renal failure two months after his admission to hospital.

Post-Mortem Examination.

At post-mortem examination fluid exuded from the incised skin, and all tissues were œdematous. The heart weighed 22 ounces. There was an area of fibrinous pericarditis over the anterior surface of the ventricles, which appeared to be dilated. In sections of the myocardium scattered areas of pallid muscle were seen to be distributed throughout.

The right pleural cavity contained a small serous effusion, the left was obliterated by dense adhesions, and the lungs were œdematous but otherwise unremarkable.

Both kidneys were of normal size. Examination of the cut surface of both revealed cortical pallor, which lay in striking contrast to the chocolate colour of the medullary pyramids.

The pancreas contained a core of yellow necrotic tissue surrounded by a zone of hyperæmia.

No abnormality was detected on macroscopic examination of the liver, spleen or alimentary tract.

Histological examination revealed the following findings. The outstanding change in all tissues was the considerable medial and intimal thickening of arteries and arterioles due to the presence of a pale, structureless collagenous material.

In the heart, areas of palely staining, disintegrating muscle fibres appeared to be related to affected branches of the coronary vessels (Figure II). There was an absence

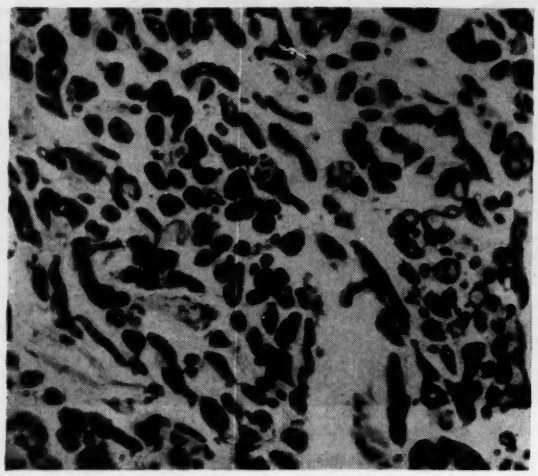

FIGURE II.
Section of cardiac muscle showing degeneration of muscle fibres. (× 275.)

of cellular infiltration, except beneath the pericardium, where there were scattered large mononuclear cells and a few polymorphonuclear leucocytes in the pericardial exudate.

In the kidneys almost complete bilateral cortical infarction was present, with only thin strands of surviving tissue extending to the capsule in association with partially occluded interlobular arteries (Figure III). In general there was a gross degree of intimal thickening with occlusive thrombosis of most interlobular vessels and the formation of mural thrombi in the yet patent larger arteries. In the surviving glomeruli and tubules there was little abnormality apart from some hyaline change in the glomerular tufts.

Beneath the thickened pleura over the lower lobe of the left lung there was extensive pulmonary fibrosis, but elsewhere the changes were again most pronounced in the vessels, and were such as have already been described in connexion with other organs.

Focal areas of infarction of the central portion of the pancreas had occurred, with a surrounding zone of vascular congestion and polymorphonuclear accumulation. An extreme degree of vascular occlusion and thrombosis was again in evidence (Figure IV).

No changes were noted in the liver apart from some cellular degeneration about the central vein of the lobules.

Discussion.

The ætiology of scleroderma is as obscure today as it was when Osler (1898) commented on the lack of knowledge as to the essential cause of this disease.

In the case reported above it is obvious that the lesions in many organs are directly related to obliterative changes in arteries and in arterioles supplying them.

Ischæmia has been put forward as the cause of the skin change by Wright and Duryee (1933). It is notable in this regard that incisions into sclerodermatous areas appear to heal rapidly, while ulcers at the tips of sclerodermatous fingers also heal after sympathectomy.

In diffuse scleroderma, the early appearance of Raynaud's phenomena in the hands, before the characteristic skin changes are obvious, not infrequently leads to an erroneous diagnosis of Raynaud's disease.

Allen Barker and Hines (1946) point out that Raynaud's disease in males is rarely encountered, although Raynaud's

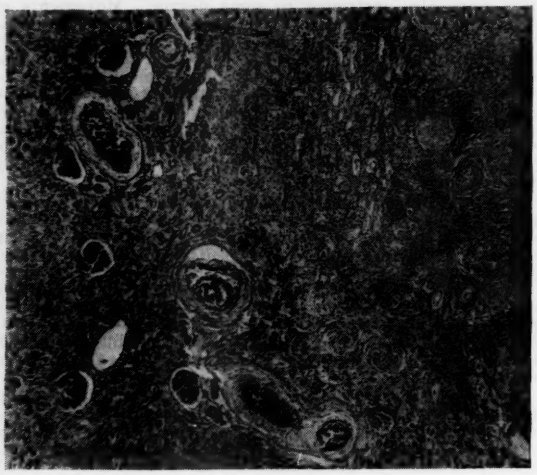

FIGURE III.
Section of renal cortex showing the focal character of the infarction, thickening of an interlobular artery and mural thrombus formation. (× 40.)

phenomena not infrequently occur in association with occlusive vascular disease, ergot poisoning, neurogenic lesions and "pneumatic hammer disease". In the case reported above, the occupational use of a pneumatic tool for some months is an interesting point. However, it is not

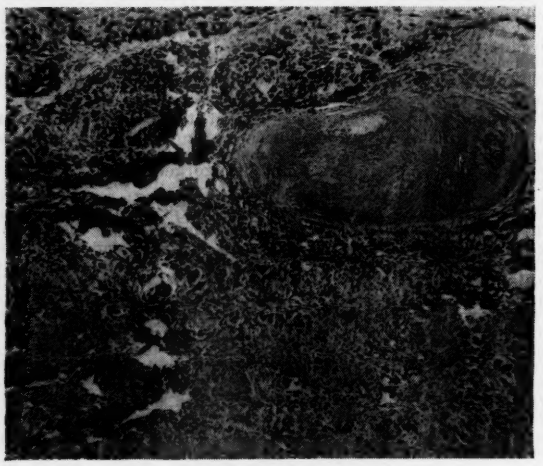

FIGURE IV.
Section of pancreas showing gross occlusive process in an artery adjacent to the infarct. (× 40.)

considered an important factor here in the initiation of the Raynaud's phenomena, in view of the constant close association of such phenomena with scleroderma.

Rake (1931) suggested as a possible cause of vasospastic phenomena in scleroderma, the degenerative changes which he observed in the sympathetic ganglia in one case. This observation does not appear to have been followed up so far by other workers.

No record has been found in the literature of patients presenting the type of pleural involvement originally shown in this case, although the appearance of a pleural effusion in terminal cardiac failure due to scleroderma is relatively common. Such massive renal and pancreatic infarctions are equally rare, although conforming to a pattern of generalized vascular disease which would lead one to expect their more frequent, if less conspicuous, occurrence in other cases of diffuse scleroderma.

Summary.

The condition of diffuse scleroderma is discussed, and a case of diffuse scleroderma with Raynaud's phenomena is reported, with post-mortem findings of widespread vascular occlusion in a number of organs, which resulted in death from cardiac and renal failure.

Acknowledgements.

We are indebted to the Chairman of the Repatriation Commission for permission to publish this case report, to Miss Helen Wischusen for the preparation of the histological sections, and to Mr. Mathaei, of the Faculty of Medicine, for the photomicrography.

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STERNO-MASTOID TUMOUR AND CONGENITAL MUSCULAR TORTICOLLIS.

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STERNO-MASTOID TUMOUR is a condition in which the sterno-cleido-mastoid muscle on one side, and very rarely on the two sides, is hard and swollen in a portion of its extent; less frequently the muscle may be involved in its entirety. Congenital muscular torticollis, expressed as an approximation of the mastoid process with the sterno-clavicular articulation on the affected side, may be found in association with the sterno-mastoid tumour. More frequently the torticollis becomes apparent only at a later period, usually at a time when growth is occurring rapidly. In many cases congenital muscular torticollis develops in patients in whom a sterno-mastoid tumour is not known to have existed previously.

The genesis of the pathological alteration which is present in the muscle in cases of sterno-mastoid tumour, or in cases of congenital muscular torticollis, is obscure. Suggestions as to causation have been plentiful. A detailed consideration of these theories need not detain us here, since they have been dealt with in detail, and their merits and demerits discussed by several authors, including Middleton, Von Lackum, Hough, and Chandler and Altenberg. Middleton's article was published in 1930, and his contribution to this subject was outstanding. He was the first to show the connexion, which is now accepted to exist, between the sterno-mastoid tumour and congenital torticollis. It was his recognition of the identical nature of the muscle pathology in these two conditions and his familiarity with the experiments of Brooks—experiments which he repeated—which led him to propound his theory upon the ætiology of these conditions. This theory has greatly influenced all subsequent discussions on this subject, and it has been widely, though not universally, accepted. Briefly, according to Middleton, the sterno-mastoid tumour and hence congenital muscular torticollis result from a temporary occlusion of the venous return from a portion of the involved muscle, an occlusion which he thought occurred during birth. This occlusion produces a venous infarction of the muscle which is followed by fibrosis; this, on the basis of the experimental work, should be just visible to the naked eye in fourteen days, and apparent on microscopic examination at eight weeks as young cellular fibrous tissue replacing the damaged muscle. Hough, writing four years later, regards Middleton's hypothesis as being the most acceptable explanation of the pathogenesis so far adduced. FitzSimmons is of like opinion, though this author considers that it is necessary, for an adequate explanation, to postulate an additional permanent element to the obstruction, and while admitting that such a permanent obstruction has never been demonstrated histologically, thinks that it may result from thrombosis of the temporarily obstructed veins.

Von Lackum, writing in 1929, states that the theory then receiving most support was that of faulty intrauterine position, and so it is interesting to note that a more recent article on this subject by Chandler and Altenberg in 1944 denotes a swing away from the theory of Middleton and a fresh emphasis on intrauterine position as a cause for the alteration in the muscle structure. Indeed, at the present time there would seem to be two schools of thought —that which accepts Middleton's hypothesis, with or without qualifications usually of a minor nature, and that which rejects this hypothesis in favour of faulty intrauterine position or of intrauterine malposition combined with trauma during delivery.

As is to be expected, the possibility that this disease may have an hereditary basis—that it may be due to some defect in the development of the fœtus—has been suggested. Middleton, and authors writing on this subject since that

time, give this possibility scant consideration. They believe that any of the facts so far recorded which seem to point in this direction cannot be excluded on the grounds of coincidence, and this may well be so. In the cases to follow there are examples of the sterno-mastoid tumour occurring in association with defects in fœtal development of a type which can be explained on an hereditary basis, or on the basis of noxious influences acting at very early stages of fœtal development. These cases will be described briefly, and only the pathological features which seem pertinent to the present study will be included. These infants died at the ages of one, three and five months respectively; the cases have been recorded in that order, since, if the muscular lesion begins to develop only at the time of birth, as has been held, then some orderly sequence in pathological progression may be apparent in the histological study of the muscle.

CASE I.—The patient was a female infant, one of twins. The twin sister is not identical; there were two placentæ and two chorions, and this twin is at the present time well and progressing satisfactorily. She presented by the vertex, and was born thirty minutes earlier than the patient, who was delivered by the breech. The patient became jaundiced on the second day, and the temperature was elevated at this time to 100·4° F. There was no evidence of a blood group incompatibility to account for the jaundice, which gradually subsided, while the red blood cells numbered 5,500,000 per cubic millimetre at the end of the first week. Though the jaundice became less intense, the infant did not thrive; she was difficult to feed and vomited frequently, and she died at the age of four weeks. She was under observation in a hospital from the time of her birth till death occurred; no tumour was ever noticed in the neck, nor was the head reported to be held in an abnormal position.

An autopsy revealed that the infant died as the result of bronchopneumonia. There was severe interstitial involvement of the lung in addition to extensive alveolar consolidation. The liver, though not enlarged, was dark green in colour, and histological examination revealed infective cholangitis; the portal tracts were liberally infiltrated with polymorphonuclear cells, and the bile ducts and canaliculi were filled with inspissated bile. The bile in the gall-bladder was viscid, but could be expressed, though with some difficulty, along the common bile duct into the duodenum. The lower half of the right sterno-mastoid muscle was swollen, hard and pale. The sternal and clavicular heads of attachment did not appear as separate tendinous structures, but merged imperceptibly with the hard swelling. A small number of red muscle fibres were spread over the surface of the swelling, while the muscle above the affected part appeared entirely normal. Additional features of note in this case were the abnormal thickness of the calvarium and the fact that the anterior fontanelle was almost closed and the sutures were united.

For histological study sections were made in the long axis of the muscle from the bony attachments below and to include a portion of the normal muscle above. In this way it was hoped that a more complete picture of the histological structure would be obtained, for in the majority of pathological studies to date which have been made from tissue removed at operation, the altered muscle is depicted in transverse section only. In these longitudinal preparations the feature which immediately attracted attention was the regular arrangement of the fibrous tissue, which replaced the muscle in the affected part. Long, thick and slightly wavy bands of collagen were progressing in the long axis of the muscle, and these bundles were composed of fine, closely packed, parallel fibrils. The nuclei, which were fairly plentiful, were extremely elongated. They ran parallel to the bundles and in many parts lay almost end to end, so that, with their cytoplasmic body, they seemed to form a complete nuclear or cellular sheath to the collagenous fibres (see Figure I). Even in an infant of this age some evidence of a tendinous attachment of the sternal head is in evidence; but in these sections no line of demarcation existed between the fibrous tissue which was replacing muscle, and what would normally be the tendinous portion of attachment, except that in the supposedly tendinous areas no remnants of muscle fibres were visible and the connective tissue nuclei were smaller and less closely approximated. Elsewhere short or longer segments of muscle fibres were present between the collagenous bundles. These fibres varied considerably in structure. Many were thick, much thicker than any fibres from the corresponding region of the sterno-mastoid on the opposite side. Others took the form of multinucleated masses as if in active growth, while still others had undergone hyaline granular degeneration. A small number of neutrophile and eosinophile polymorphonuclear cells were clustered about these degenerated or degenerating fibres, and small, darkly staining elongated nuclei were numerous at the periphery of the granular debris.

CASE II.—The patient was a female, who died at the age of three months. She was born prematurely at eight months and weighed four pounds two ounces at birth. She did not progress satisfactorily and suffered from repeated attacks of vomiting and diarrhœa. She was constantly under observation in hospital, but no tumour or other abnormality of the neck was ever observed.

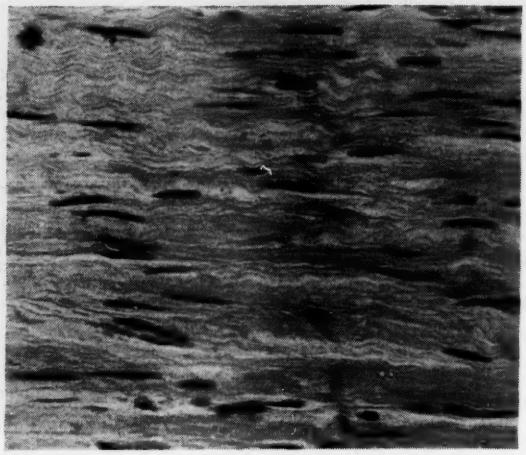

FIGURE I.

Fibrosed portion of right sterno-mastoid muscle cut in the long axis. The muscle is replaced by bundles of collagen fibres which run parallel to one another and to the elongated connective tissue nuclei. (Hæmatoxylin and eosin stain × 450.)

Autopsy revealed that the infant died as the result of renal failure. The kidneys were extremely small and weighed 3·5 grammes and 3·3 grammes respectively. Histological examination revealed a similar defect in each kidney. The architecture of the kidneys was distorted; glomeruli were scanty and the cortical tubules were dilated, so that small cysts of varying size had developed (see Figure II). Small islands of cartilage are a frequent finding in these malformed hypoplastic kidneys, and such islands were present in this case.

The lower half of the right sterno-mastoid was enlarged, extremely hard, pale and glistening, and this alteration extended from the points of attachment to the sternum and clavicle to a point midway up the muscle belly. The muscle above the swelling was normal, as was the sterno-mastoid muscle on the left side.

In this case also, blocks were taken in the length of the involved muscle, and the histological changes found were so similar to those recorded in Case I that no lengthy description of them need be given. The regular arrangement of the replacing fibrous tissue was once again apparent. There were no fibres crossing the main bundles obliquely or at right angles. Short muscle fibres showing reparative or degenerative changes were present amongst the connective tissue, while the degree of cellularity of this fibrous tissue appeared to be neither more nor less than that seen in Case I. The only additional feature of interest was pronounced *osteitis fibrosa*. This had resulted from hyperplasia of the parathyreoids and was secondary to the renal failure.

CASE III.—The patient was a male infant, who died at the age of five months. It had been noticed that this infant held his head flexed to the left; but little regard had been paid to this abnormality, because it was overshadowed by a severe illness, which began at the age of six weeks, and which was characterized by considerable respiratory obstruction and a pronounced expiratory wheeze.

Autopsy revealed obstructive pulmonary emphysema. The trachea and bronchi were filled with a tenacious purulent exudate, while in the lung substance there were many large red to yellow foci of bronchopneumonic consolidation. The pancreas was finely granular and slightly smaller and

firmer than normal. Histological examination of this organ revealed the typical alterations of structure seen in pancreatic steatorrhœa or so-called cystic fibrosis of the pancreas. These alterations will be appreciated by reference to Figure III, in which the distended ducts and acini can be seen, filled with inspissated secretion. The entire left sterno-mastoid muscle, from the mastoid process to the

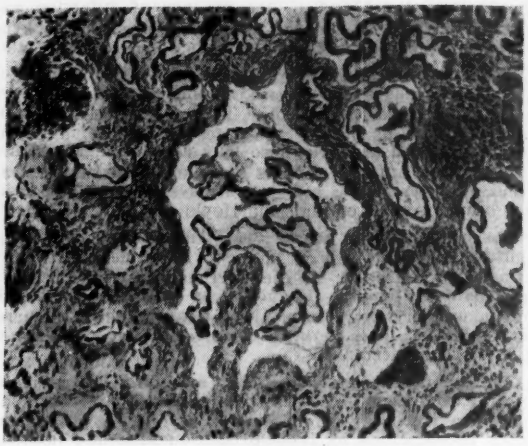

FIGURE II.

Cortex of hypoplastic kidney. There is complete disorganization of the renal architecture, which is replaced by small cysts. (Hæmatoxylin and eosin stain × 100.)

sterno-clavicular attachment, was replaced by a thick white cord of cartilagenous firmness. This cord did not present the usual almost strap-like flattened shape of the sterno-mastoid muscle, but was almost rounded, except above, where it became flattened near the mastoid attachment. Below, the rounded body divided into two heads of insertion,

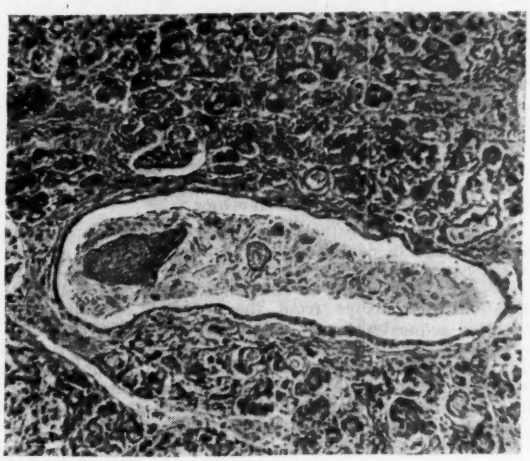

FIGURE III.

Centrally situated is a distended pancreatic duct containing inspissated secretion and desquamated lining cells. Atrophy and fibrosis are evident in the surrounding glandular tissue and the acini contain inspissated secretion. (Hæmatoxylin and eosin stain × 100.)

sternal and clavicular, and no distinction could be drawn between the appearance of these heads and the body of the muscle.

In sections cut longitudinally from the muscle in this case, the histological appearances closely resembled those seen in Cases I and II. In the lower third the muscle was entirely fibrous, no muscle fibres remaining; in the upper two-thirds, however, muscle fibres of varying size were present and showed regenerative and degenerative alterations. The cellularity of the fibrous tissue in this case did not appear to be less than in Cases I and II.

Except in Case I, in which a small number of hæmosiderin-laden histiocytes were found about some of the vessels, evdence of past hæmorrhage into these muscles was entirely lacking, and in no case did the blood vessels or nerves show any pathological change.

The most recent example of sterno-mastoid fibrosis to come to autopsy was discovered after the above-mentioned cases had been collected and described, and for the sake of reference this case will be referred to as Case IV.

CASE IV.—The patient was a female, who died at the age of six weeks. In this infant the medial half of the left sterno-mastoid, from the sterno-clavicular attachment to the mastoid process, was replaced by fibrous tissue of dense consistency. Histologically this fibrous tissue resembled that found in the three cases above described. In addition to the muscular abnormality multiple congenital defects were present in this case. The central nervous system was extensively malformed. There was a large lower thoracic and lumbar myelomeningocele with an associated though atypical Arnold-Chiari malformation. The left lobe of the cerebellum had failed to develop, the ventricles of the cerebrum formed but a single cavity, and all the cerebral convolutions were extremely small. The urinary tract, too, was malformed. In the absence of any cause for anatomical obstruction the bladder was thick-walled, the ureters were hypertrophied though not dilated, and there was appreciable bilateral hydronephrosis.

Discussion.

Though the ætiology of congenital muscular torticollis remains obscure, at least certain features of the disease may be accepted as established. The condition is by no means rare and the incidence has been variously estimated as ranging between 0·3% and 0·5% of all births. There is an equal sex incidence and no predilection for any race. Bilateral involvement of the muscles is rare, but the lesion occurs with about equal frequency on the right and the left sides. Middleton states that the tumour is never present at birth; Chandler and Altenberg, on the other hand, maintain that the tumour may be present at birth or may become manifest only about the tenth to the fourteenth day. These latter authors believe, from their experience based upon the study of 101 patients operated upon at ages varying from three to four weeks, that the swelling increases in size for two to four weeks after birth and then begins to regress, and may disappear, as a swelling, in from five to eight months. That a tumour may be present and escape recognition is apparent and is sufficient explanation of the large number of cases of congenital muscular torticollis developing months or years later, in which no history of any previous abnormality can be obtained.

In all the theories propounded none can give an adequate explanation of the curious fact that of all the muscles of the body the sterno-mastoid should be unique in undergoing partial or complete fibrous replacement from birth. Perhaps a similar abnormality is the cause of some cases of congenital high scapula—Sprengel's deformity. This curiously unique quality of the lesion cannot fail to attract attention, and in this connexion the writer has recently had the opportunity to study tissue removed from the *rectus abdominis* muscle of a young infant in whom the entire right rectus muscle and the upper portion of the left rectus muscle were dense and white and of a tendinous consistency. Histological examination revealed fibrous replacement of the muscle, with regularity of arrangement of the fibres and a general structure closely resembling the lesion of the sterno-mastoid. There was nothing to suggest that trauma had played a part in this case. That the two conditions, one affecting the sterno-mastoid and the other the *rectus abdominis*, are related cannot be determined by resemblances in histological structure, but such a possibility would seem to exist. It is difficult to understand how faulty intrauterine position of the fœtus could be supposed to produce an abnormality such as that found in this rectus muscle.

Evidence so far adduced for an hereditary basis is scanty, but not entirely lacking. In the 101 cases studied by Chandler and Altenberg, associated abnormalities capable of detection by clinical methods were of low incidence. Erb's palsy was an associated condition in three cases, there was hypertrophic pyloric stenosis in three cases and a congenital short arm was present on the involved side in one case. Further, two mothers had untreated muscular torticollis and the brother of one patient had a fibrous cord replacing the sterno-mastoid but no muscular torticollis. Von Lackum reports three of four children in one family who had congenital muscular torticollis. That associated congenital abnormalities sufficient in degree to cause death in infancy are uncommon, is suggested by the fact that a search of the literature, though admittedly incomplete, failed to reveal any reports dealing with autopsy material; the reports were concerned solely with material obtained at operation. It is true that the simple association of sterno-mastoid fibrosis with other anomalies and abnormalities of development in no way proves a common origin for the two. Moreover, in the cases reported here the material is obviously selective; the associated congenital defects were the ultimate cause of death in each of the four examples, and these were the only examples of this condition found in a series of 2000 complete autopsies performed on infants and children.

Conclusion.

Three features suggest that the current ideas concerning the formation of the sterno-mastoid tumour and subsequently of sterno-mastoid fibrosis are not entirely acceptable:

1. The possibility, from reports in the literature and from the experience of the cases reported here, that an hereditary fault in the development of the muscle *Anlage* may be responsible for the condition.

2. The tendinous appearance of the altered muscle both on gross and on microscopic examination; the fact that discrete "scars" have not been found in the substance of the muscle, and that the altered muscle seems to merge with the tendinous insertions, suggesting an overgrowth of tendon at the expense of muscle.

3. The inability of any suggested methods of causation to explain adequately the development of a fibrous mass in the sterno-mastoid muscle of a type not known to occur in any other muscle in the newborn infant.

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SOME ASPECTS OF PREVENTION AND TREATMENT OF BRONCHIECTASIS.[1]

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No one who has postured a patient with bronchiectasis can fail to be appalled at the nature of this distressing condition. To see a young and beautiful child or adolescent open her mouth to let flow six to eight ounces of foul pus is one of the more unpleasant experiences of medicine. It is incumbent upon us all, in this age of prophylaxis, to do what we can, not merely to treat the disease when established, but more particularly to prevent its occurrence if that is possible.

Bronchiectasis is by definition dilatation of the bronchi. It is, as Lander and Davidson (1938) pointed out, essentially a morbid anatomical change dependent on some antecedent pathological event, and should not be regarded in the earlier stages and in the absence of infection as a disease in itself. It does not necessarily produce any symptoms, but apparently lessens the functional capacity of the bronchi to expel any sputum which may be produced.

If infection is superimposed, secondary changes are produced in the dilated bronchi. These result in most cases in production of pus by the infected mucosa, a process accelerated and aggravated so long as sputum is retained. In a smaller number of cases granulations only result and recurrent hæmoptyses are the only symptoms. This is the dry bronchiectasis of Wall and Hoyle (1933).

These secondary changes are manifested by cough, sputum and fever. The patient is liable to recurrent attacks of infection in the surrounding lung tissue or pneumonitis, usually diagnosed as pneumonia, bronchopneumonia or severe bronchitis. He may have recurrent hæmoptyses. These clinical manifestations make up the disease bronchiectasis.

The symptoms may arise after some illness or incident or may commence insidiously. The illnesses most frequently noted are whooping-cough, measles, diphtheria, pink disease when accompanied by bronchitis, bronchitis and influenzal bronchopneumonia, all conditions in which the patient produces viscid sputum in his lungs, and many of them conditions in which there is some degree of bronchial spasm. They are also all infections, but there is no organism common to them all.

Prevention of bronchiectasis must be based on some understanding of the ætiology and pathogenesis.

Dilatation of the bronchi occurs when there is a persistent block or obstruction of a bronchus together with infection, as in carcinoma or adenoma of the bronchus, or foreign body. It may arise after lung abscess. This type is secondary bronchiectasis, and the primary condition is the one on which most attention is focused. It may occur in tuberculosis owing to stenosis of bronchi by fibrous tissue or by pressure of enlarged lymph nodes.

Prevention in this variety lies in early removal of foreign bodies; in the early treatment of lung abscess while it is in the acute or subacute stage (that is, in the first six weeks), which, as Neuhof and Touroff (1938) have demonstrated, results in healing without secondary changes in the lung; in early treatment of tuberculosis before chronic fibroid tuberculosis develops.

Primary bronchiectasis may be congenital or acquired. It may occur in conjunction with developmental anomalies of the heart and great vessels, as in *situs inversus*, and in errors in blood supply to the lungs, as when the lungs are supplied by arteries from the systemic circulation and so-called sequestration of the lung occurs. On the facts at our command at present there is no way of preventing this type of bronchiectasis, though it is possible that further studies along the lines that have been so fruitful in elucidating the ætiology of congenital heart disease may provide the answer in these cases.

In the so-called congenital cystic bronchiectasis, dilatation of the bronchi is seen together with gross distention of the bronchial tree and cysts throughout the lungs. The cysts may or may not communicate with the main bronchi. The cavities are lined by epithelium columnar or cubical, often ciliated, and there is gross irregularity in the arrangement of the structures in the walls of the cavities and dilatations. Norris and Tyson (1947) have described two cases clearly of this type, and suggest that the development of the condition is similar to the development of congenital cystic disease in the kidneys, liver and pancreas. Infection usually supervenes on this condition and so alters the lung that it may be difficult to place a case with infection in its correct category. This group is similar to the last in regard to prevention.

[1] Read at a combined meeting of the Section of Surgery, the Section of Anæsthesia and the Section of Pædiatrics, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

There remains the vast majority of cases in which dilatation of the bronchi occurs—the common or acquired type of bronchiectasis.

There are divergent opinions as to the ætiology and pathogenesis of this type of bronchiectasis; but there is no doubt that at least two factors are involved. The divergence is as to which is the primary. These factors are infection and atelectasis. Pathological material which has been studied has been derived from two sources, the post-mortem room and the operating theatre. The former has received attention from many famous pathologists for very many years. The latter has been available only in the last twenty years. There is no doubt that this operation material represents the condition as it exists in the living, and on it the pathology of the living may be studied. Autopsy material is either bronchiectatic tissue from patients who have died of the disease or its complications, and therefore represents the end result of the pathological process, or, in a smaller number of cases, bronchiectatic tissue which has been present coincident with some other condition which has killed the patient.

More respect should be paid to the lessons learned from the operation material than to the autopsy, and many of the more ancient views of the pathology of bronchiectasis should be discarded.

Investigation of the surgical material shows that atelectasis is present in a large majority of cases, up to 80% in some series (Mallory, 1947). In most there is some infection. However, the changes in the bronchi vary greatly; but in nearly all the outline of the normal arrangement of the several layers of the bronchial walls may be seen. Inflammatory changes may extend through the walls of the bronchi to the surrounding lung tissue, but in the main the changes are in the walls of the bronchi in the form of infiltration by fibrous tissue. Masses of round cells and many lymph follicles may be seen. Where there has been gross clinical infection, little normal lung tissue may be present.

The protagonists of the infection theory of bronchiectasis hold that the infection in these cases—whooping-cough *et cetera*—has, in some way unspecified, caused a weakening of the bronchial walls; some even hold that the infection has caused excavation of the lung, and that the excavated areas then become lined by elements similar to those which line the smaller bronchi, so that the bronchiectatic dilatations are in fact excavations communicating with bronchi (Lisa and Rosenblatt, 1943).

Others believe that the infection merely weakens the walls, and the mechanical arrangements within the chest are such that the bronchi become dilated as a result of frequent increases of stress during coughing and effort, or by reason of the weight of retained secretion. One obvious weakness in this theory is that no specific type of infection is responsible—in other words, almost any type of infection can sometimes weaken the walls of the bronchi, but nothing is known as yet as to why the weakening occurs only in some cases and at some times, and not always.

On the other hand, all the patients whose bronchiectasis has had a definite onset have suffered from a disease in which there was inflammation of the walls of the bronchi with production of sputum, and in many cases spasm of the bronchi; so long as this is true, then these patients have had in their bronchi viscid sputum which could act as a plug and obstruct a bronchus.

Atelectasis is common in the post-operative course of many conditions, particularly those in the upper part of the abdomen, in which adequate movements of the diaphragm are restricted by the incision in the abdominal wall, and in which there has been some post-operative inflammation of the bronchi. It may occur after operations on the upper part of the respiratory tract, and post-operative atelectasis is common after lobectomy. Bronchoscopic examination in such cases shows that the cause of atelectasis is obstruction of a bronchus by blood, debris, a plug of sputum or even a film of mucus no thicker than a soap bubble stretching across the mouth of a bronchus. This happened in one of my cases, in which the film was so nebulous that it was scarcely credible that it could in fact have blocked the main bronchus, but as it was sucked away movement of the chest wall on the side of the collapse was reestablished. X-ray examination confirmed reexpansion of the lobe (Case II).

If this can happen in this type of case, it is certain that it can and does happen in any case in which viscid sputum is present in the bronchi, and the only problem is why it does not occur more often. Once atelectasis has occurred, that portion of the lung is much smaller than it should be and occupies less space. This results in increased negative pressure in that side of the thorax. The diaphragm moves up, the mediastinum moves over, and the remaining non-atelectatic lung expands in response to this increased negative pressure. The effect within the bronchi is that the force which acts on the plug and thrusts it down the bronchus is greater by the amount of increased negative pressure which remains, and so the block is pushed further and further into the bronchus. This is certainly the case when the plug has been formed by a foreign body such as a peanut, and all bronchoscopists have commented frequently since Chevalier Jackson's pioneer observations that the peanut goes further and further down the bronchus and becomes less and less visible.

It is obvious that when all attempts at removal of such a foreign body by bronchoscopy have failed, a natural step is to diminish the negative pressure in the chest by the injection of air in the form of a pneumothorax. This diminishes the negative pressure in the thorax and diminishes the force which acts on the plug and pushes it down the bronchus. Not infrequently the patient can then expel the plug.

Thus, whatever one's views on the pathogenesis of bronchiectasis, the conditions necessary for the production of atelectasis are present in the infection which heralds the onset of bronchiectasis in so many cases, and it is difficult to escape the conclusion that atelectasis is the factor which occurs only sometimes, and this explains the absence of specific organisms and why bronchiectasis develops only sometimes.

The studies of Habliston (1928) and others show that if atelectasis is present there is increased negative pressure in the pleural cavity in these circumstances, and bronchograms reveal that the bronchi are dilated. Further, when artificial pneumothorax was used as a method of treatment for bronchiectasis, the introduction of air into the chest, which diminished the excess negative pressure, decreased the size of the dilated bronchi.

As atelectatic lobes may reexpand, it is obvious that the bronchi must return to normal, and bronchograms show that this is so.

Moreover, reexpansion is possible for some time, and we have X-ray films from a case in which this has in fact occurred (Case III). As this patient had a cough and much purulent sputum over many months, she did in fact have the "disease bronchiectasis".

The patient, a girl, aged ten years, had had a cough and sputum for many months, the latter ranging in amount up to half a cup and being foul and thick. A bronchogram of the left side in September, 1949, revealed dilatation of the left lower lobe bronchi, together with crowding of the bronchi and atelectasis of the lower lobe of the left lung. Her apex beat was in the fifth intercostal space, one inch below the nipple. There was flattening of the left lower quadrant of the chest, with some sternal retraction on inspiration. Harsh breath sounds and rhonchi were heard at the base of the left lung in the axilla posteriorly. Postural drainage was instituted with considerable diminution of cough and sputum. In December a bronchogram of the right side was made; this caused the child a fair amount of upset, and for a week or two she had a great increase in her cough and sputum. Two weeks after the second bronchogram her cough and sputum disappeared, and the mother said that the patient was better than she had ever been in her life. In January she still had harsh breath sounds. In March the child looked very well, she had had no cough or sputum for some two months, the flattening of the left side of the chest wall was much less, and the apex beat was now in the fourth intercostal space just below the nipple. Though there was still some sternal retraction on inspiration, it was very much less than formerly. The breath sounds were now normal,

and no adventitiæ were present. A bronchogram made at this time showed that the bronchi were now similar to those on the right side, they were not dilated, and they were not crowded. There was no atelectasis, and the lingular bronchi and the bronchi of the upper lobe, which formerly had been kinked downwards owing to the over-expansion of the upper lobe, were now back in their normal positions.

For such a reexpansion to occur the bronchial walls must still be reasonably normal. One cannot conceive of reexpansion of a bronchus which is infiltrated with fibrous tissue and round cells or surrounded by fibrosed lung tissue, and so this must represent a stage in the development of bronchiectasis as seen in the post-mortem room in many cases, if not in all; probably this is so in all cases in which atelectasis is present. Investigation of surgical material shows that such cases are the majority, and in fact the only cases of bronchiectasis in which it cannot play a part are those in which bronchograms reveal no crowding of the bronchi. In cases in which atelectasis is present, then infection would appear to be the second event; but that does not lessen its importance, because it is the changes produced by infection which render the condition irreversible.

It is worthy of note that cough and sputum and fever due to bronchitis often increase in amount when atelectasis occurs in post-operative cases, and that reexpansion of the lung can be diagnosed just as surely by the disappearance of the cough and sputum and fever as by the X-ray findings. This is seen not only in atelectasis after abdominal operations, but also in the post-operative atelectasis after lobectomy and thoracoplasty. Further, if an underwater drain is used in these cases, increased negative intrapleural pressure is a sure indication that atelectasis has occurred.

Another patient (Case IV), a male, aged twenty-two years, in August, 1948, developed right pneumonia and pleurisy and empyema after a tooth extraction.

On his admission to hospital the left side of his chest was clear on clinical and radiological examination. The empyema was drained; prior to closure of the cavity the patient became febrile coincidently with a block in the drainage tube. The next X-ray film showed collapse of the lower lobe of the left lung. He had no symptoms from this, and the empyema cavity closed and he was discharged from hospital in December.

In January he was readmitted to hospital with left lower lobe pneumonia and a slight effusion. He recovered quickly, but then began to cough and raise copious sputum. A bronchogram revealed gross bronchiectasis of the lower lobe of the left lung and the lingular process, but the bronchi of the rest of the upper lobe were normal in size. He later developed an empyema on the left side. This was drained, and he then produced about 10 ounces of sputum, and there were about 12 ounces of purulent discharge from the empyema. He had no bronchial fistula. The empyema cavity showed no sign of diminution in size, and his condition remained unchanged until July. X-ray films showed that the upper lobe was clear, but then within a week the upper lobe became opaque and remained so despite bronchoscopy. Soon cavities could be seen in the upper lobe. At pneumonectomy in November (delayed by an attack of infectious jaundice), gross bronchiectasis was present in all bronchi of the left lung, and in the lower and lingular lobes the bronchiectasis seemed worse than when the bronchograms were taken.

Thus in this case atelectasis of the lower lobe of the left lung occurred without cough or sputum and preceded the bronchiectasis. Atelectasis of the upper lobe preceded the bronchiectasis in this lobe also. (It might be argued that, as there was a tube in the empyema cavity, the necessary excess negative pressure would not develop, but the thick wall of the empyema cavity would be rigid and the collapse of the upper lobe could still create an excess negative pressure on the lung side of the thickened pleura.)

That bronchiectasis may spread when there is much sputum has been sufficiently demonstrated in many series. The explanation of this phenomenon is of great importance, because if it is accepted that the condition is progressive, prophylactic operation is indicated. But is there any other explanation? Undoubtedly so long as cough and sputum are present, further bronchial obstruction may occur with further atelectasis, infection and bronchiectasis. But whether such a spread occurs if adequate drainage is used is another thing entirely.

Consideration of the facts so far adduced suggests that there is a field for the prevention of bronchiectasis, firstly by the prevention of the infectious diseases which appear to be associated with the onset of bronchiectasis, secondly by adequate supervision in such cases when they occur. McNeill (1932) made the following statement, which is just as true today:

> But it is a known and striking fact that although these diseases in their severest forms are very numerous in hospital practice and under close observation, bronchiectasis is seldom detected there in its earliest stage. The symptoms of the condition thus do not seem to start at full blast.

The atelectasis that may have occurred gives rise to few symptoms in the child, and as a residual cough is common and the child swallows the sputum, the condition is not diagnosed; but an X-ray examination will reveal any atelectasis, and this is the time to find it, when it is still reversible.

I have indeed endeavoured to investigate on these lines all patients admitted to hospital with whooping-cough. Unfortunately, owing to administrative difficulties, the investigation has not been as thorough as I would wish. What I desire to do, and what I suggest should be routine practice everywhere, is that all patients suffering from whooping-cough, measles and diphtheria should be radiologically examined on their admission to hospital, either *en route* to the ward or as soon as they are in bed, that they should be radiologically examined at any time when the course of the disease ceases to be normal, and that they should be radiologically examined on their discharge from hospital. If there is any change in the X-ray findings, the patient should be subjected to bronchoscopic examination. I have failed to secure these necessary X-ray examinations in all cases of whooping-cough, but such X-ray films as I have been able to obtain show that in some cases there is an area which could be read as atelectasis, and in others there are patches of consolidation together with hilar opacities suggestive of enlargement of the lymph nodes of the hilum (Cases VI, VII and VIII).

I believe, therefore, that in these cases atelectasis may occur owing to obstruction of a bronchus by a plug of sputum, and that atelectasis may occur owing to an obstruction of the bronchi by the pressure of lymph nodes in the hilum of the lobe, enlarged as a result of infection in the lung.

This state of affairs was found in a lobe, removed from a child, aged twelve years, in which the bronchi were much narrowed towards the hilum by enlargement of lymph nodes outside, so that the point of the scissors could be inserted into the bronchi only with great difficulty. Beyond this area the bronchi were dilated mildly and contained pus, and at the periphery there were several small saccular dilatations.

Further, I believe that in all such cases in which there are X-ray changes, bronchoscopic examination should be made and the bronchi cleared if possible.

I believe that all patients with such infectious fevers should receive antibiotics to reduce the incidence of secondary infection of the bronchi and thus diminish the number in which enlargement of glands will occur.

All patients with bronchitis and pneumonia should be radiologically examined. (Such X-ray films must, of course, be in two planes, postero-anterior and lateral.) If opacities persist after ten days a bronchoscopic examination should be made. Pathologists who teach that there is a condition of unresolved pneumonia have been responsible for the failure to attack the illness when treatment is still possible.

I believe that this course should be followed in all infectious diseases associated with bronchitis. I believe that it should be followed in all cases of pink disease in which bronchitis is present and in which the X-ray findings are not normal when the child is ready for discharge from hospital. I believe that all patients who have undergone tonsillectomy or other operations on the upper part of the respiratory tract, and in whom a cough develops after operation, should be radiologically examined and if necessary submitted to bronchoscopic examination. I believe that all patients who have post-operative pulmonary complications should be radiologically examined, that they should not be discharged from hospital until an X-ray

film shows that their lungs are normal, and that bronchoscopic examination should be performed if necessary.

This programme sounds a very ambitious one; obviously many more X-ray examinations must be made than are being made at the moment. Nevertheless, at a time when the public are prepared to have mass radiology for pulmonary tuberculosis, it would seem that this field is a much more profitable one than that of tuberculosis, for if these residual pulmonary complications are picked up at a stage when the dilatation of the bronchi is still reversible, then these patients can be returned completely to normal; moreover, the incidence of this disease, which in its manifestations is only less horrible than tuberculosis by reason of the fact that it is not infectious, can be greatly diminished.

Treatment.

The disabilities produced by bronchiectasis are cough and offensive sputum, recurrent hæmoptysis and recurrent attacks of inflammation in the lung surrounding the dilated bronchi, and the possible development of lung abscess.

Eradication of the disease when possible is, therefore, the ideal treatment.

Other methods of treatment at our disposal are based on the principles of adequate drainage of the dilated bronchi and reduction of infection. With these methods all patients can be improved in health and some may be rendered sputum-free so long as the treatment is continued. Adequate drainage is secured by postural drainage of the affected bronchi. The drainage must be carried out for an adequate time, at least all night and three times during the day, this being preceded by a period of about one month during which continuous drainage is maintained for at least twenty-two hours a day.

After postural drainage, then the use of antibiotics by inhalation *et cetera* may further reduce the sputum or the organisms therein; but no treatment directed at the infection only can secure relief of symptoms unless adequate drainage is also secured.

It is useless to expect that the intermittent postural drainage introduced by Laennec one hundred years ago and carried out for three or four periods a day, of twenty minutes each, will empty the bronchi and keep them empty; nor can this condition be achieved by an occasional bronchoscopic examination. Patients who have had frequent bronchoscopic aspirations and antibiotics and then have had continuous postural drainage are emphatic that the latter treatment is infinitely better.

Further, it is probable that if a patient can be rendered sputum-free by drainage compatible with a reasonable life, or if the sputum can be reduced to one or two drachms, conservative treatment should be persisted with. If not, then radical treatment should be instituted when possible.

Eradication of the disease is possible by surgical operation of lobectomy (segmental or lobar) or pneumonectomy. During the operation the patient depends on the other lung for his respiration. It follows that the disease must therefore be confined to one lung or to such portions of both lungs that existence is possible on what remains both permanently and during the second operation. This amounts to bronchiectasis which involves two lobes on one side (one of which must be the lingular or middle lobe) and one lobe on the other side, or less than this.

Although operation for bronchiectasis now carries a low mortality rate (1% to 2%), yet there are some qualifications as to its success. Firstly, some patients have persistent mucous sputum, though no pus. Some may develop a bronchial fistula, though the incidence of this is lessening; occasionally in conjunction with the latter reexpansion of the remaining lobe may not occur, and if infection is present will necessitate removal of the remaining lobe.

However, if all the diseased tissue is removed at operation, the patient will be completely cured in the absence of the above-mentioned complications.

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Reviews.

DISEASES OF CHILDREN.

BRUCE WILLIAMSON'S compact "Handbook on Diseases of Children" has recently reappeared in a sixth edition which is practical evidence of its popularity and usefulness.[1]

This little book is well printed and classified for ready reference by the student or the busy practitioner. It has been brought up to date conservatively to include many of the recent additions to pædiatric knowledge. The illustrations and diagrams are of a high order and embellish the text, but they also epitomize the information and engage the reader's attention.

It is of interest to record as an instance of the inclusion of recent material that there are references to cortisone and ACTH therapy for rheumatic carditis and for juvenile rheumatoid arthritis and it is timely to give wide publicity to the dangers of these new agents. Williamson states that wounds fail to heal, granulation tissue does not develop, serious derangement of adrenal function with obesity and hypertension has been observed and healed tuberculous lesions may become unsealed.

Quite properly emphasis has been placed on the preventive aspects of the medical care of children with particular attention to the period of early infancy. The mortality returns of the Registrar-General for 1947 are used to punctuate the improvement of the survival and morbidity prospects during the past forty years.

In many places Williamson writes enough for the reader to want to know more, but we think that the weight of a handbook should not tire the hand and those in search of specific facts can readily seek them elsewhere.

ATLAS OF SKIN AND VENEREAL DISEASES.

THE book by Frieboes and Schönfeld is an atlas of convenient size, beautifully edited.[2] The excellence of the coloured reproductions and black-white illustrations makes it of oustanding value to general practitioners and teachers. It also contains much that is of interest to specialists. The letterpress is in the German language.

Despite the fact that this book is the work of two authors—one of them, Professor Frieboes, is unfortunately dead—it is a model to other writers. The authors have succeeded in maintaining a very high standard of German, free of the bombastic and propagandist phraseology which was so popular even with the scientific literature during the period of Nazi domination.

[1] "A Handbook on Diseases of Children: Including Dietetics and the Common Fevers", by Bruce Williamson, M.D. (Edin.), F.R.C.P. (London); Sixth Edition; 1951. Edinburgh: E. and S. Livingstone, Limited. 7¼" × 5", pp. 454, with many illustrations. Price: 17s. 6d.

[2] "*Atlas der Haut- und Geschlechtskrankheiten*", by Dr. Walter Frieboes and Dr. Walther Schönfeld; 1949. Stuttgart: Georg Thieme Verlag. 11" × 8", pp. 276, with 428 coloured illustrations. Price: D.M.66.

The advanced student of dermatology will appreciate the comprehensive nature of the work. He will also find the many references to treatment extremely helpful and convenient. The photographs, of which there are more than 400, deserve special attention, for they are well chosen and beautifully reproduced. The atlas is printed on a high quality paper.

Australian readers will be interested in descriptions and reproductions of conditions rare in this country, for example, atrophy and tuberculosis.

There are, of course, some omissions. For example, sulphapyridine finds no mention in the therapy of *dermatitis herpetiformis*. Psychosomatic features in the genesis of skin diseases are not stressed.

A mild protest must be made against the use of an obsolete adjective "ruber" in connexion with *lichen planus*. *Lymphogranuloma inguinale* of the German authors is generally called *lymphogranuloma venereum*. The view of the importance of dermatograms for the diagnosis can hardly be shared.

We think that in the next edition the following additional pictures should be included: (i) Very common, but from medico-historical point of view interesting, tattoos produced on the arms of inmates of German concentration camps. (ii) Tropical lichenoid dermatitis caused by "Atebrin" because of its tendency to simulate other dermatoses. (iii) *Balanitis xerotica* Stühmer. (iv) *Granuloma inguinale*, "the fifth venereal disease".

The slight criticisms apart, the authors and publishers are to be congratulated on producing a very useful book which should have a ready sale.

ANKYLOSING SPONDYLITIS.

JOHNSON AND LAW'S well-printed, well-illustrated and extremely readably written book deals with a subject which is progressively finding its way into the journals of the world.[1] Surprisingly little has been written in book form regarding ankylosing spondylitis, and as the authors point out, the only other existing volume is the original monograph by Dr. Gilbert Scott. Being written by a radiographer with a very considerable clinical experience and a surgeon, the book covers a wide approach to this debilitating syndrome. A very striking and informative synthetic history is given in the early pages describing the progressive stages of a disease, which if dealt with according to this book's very practical approach should cease to be a major form of crippling and invalidism. It is a striking fact that ankylosing spondylitis attacks the young, the healthy and the athletic. The tremendous importance of early diagnosis is stressed in this readable book which contains a vast amount of information in its 184 pages. It is a book which should be on the shelves of every practitioner.

PERORAL ENDOSCOPY.

THE name of Chevalier Jackson immediately calls to mind peroral endoscopy. He it is who has been responsible for much of the pioneer work in this field; moreover, his writings on the subject have always been as comprehensive as they have been authoritative. (As proof of this, in a bibliography of 429 references in the present volume, 223 refer to Chevalier Jackson.) Not only was he one of the first to use the bronchoscope in its present form at the turn of this century, but also a few years later, in 1907, he wrote the first textbook on the subject, and later still in 1920 was appointed professor to the first chair of bronchoscopy and œsophagoscopy—qualifications warranted to stamp with authority any writings on the subject.

In "Bronchoesophagology" father and son (Chevalier L. Jackson) have again collaborated.[2] This work supersedes their text-book on bronchoscopy and œsophagoscopy and adds current concepts and newer developments. Two-thirds of the book are devoted to bronchology; the remainder is concerned with œsophagology. Anatomy, instruments, techniques and appearances are all carefully presented. The outstanding merit of the book, however, lies in the detailed chapters on foreign bodies and on obstructive conditions of the bronchi and œsophagus. The format is faultless. The legends to the illustrative sketches and plates are so lucid as to make the text almost superfluous. We should indeed be grateful for such outstanding ability to pass on knowledge and methods by means of such excellent books as this their most recent.

The Jacksons' methods have received world-wide acclaim. This is not to be wondered at when they have reached a point where they can pronounce that 99% of inhaled foreign bodies in their clinic have been successfully removed.

This book is a necessity for those concerned with diseases of the air and food passages. It must surely become the standard reference work in this field.

[1] "Ankylosing Spondylitis: A Practical Guide to its Diagnosis and Treatment", Part I, by F. Hernaman-Johnson, M.D., F.F.R., D.M.R.E., Part II, by W. Alexander Law, O.B.E., M.D., F.R.C.S.; 1949. London and Australia: Butterworth and Company (Publishers), Limited. 8½" × 5½", pp. 218, with 41 illustrations. Price: 34s.

[2] "Bronchoesophagology", by Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., and Chevalier L. Jackson, M.D., M.Sc., F.A.C.S.; 1950. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 7", pp. 380, with 260 illustrations. Price: £5 18s. 9d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Microkaryocytes, the Fourth Corpuscles and Their Functions", by K. G. Khorozian, A.B., M.S., M.D.; 1951. Boston: Meador Publishing Company. 8½" × 5½", pp. 970, with 255 figures. Price: $12.00.

The author claims to have made a remarkable discovery.

"Pediatric Allergy", by Robert Chobot, M.D.; First Edition; 1951. New York: McGraw-Hill Book Company, Incorporated. 8" × 5¾", pp. 294. Price: $4.50.

Intended for the student taking a course in pædiatric allergy, for the pædiatrician and for the general practitioner.

"The 1950 Year Book of Neurology, Psychiatry and Neurosurgery" (November, 1949-October, 1950): Neurology, edited by Roland P. Mackay, M.D.; Psychiatry, edited by Nolan D. C. Lewis, M.D.; Neurosurgery, edited by Percival Bailey, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 628, with 121 illustrations. Price: $5.00.

One of the Practical Medicine Series of year books.

"A Syllabus of Laboratory Examinations in Clinical Diagnosis: Critical Evaluation of Laboratory Procedures in the Study of the Patient", edited by Thomas Hale Ham, B.S., M.D.; 1950. Cambridge, Massachusetts: Harvard University Press. 11" × 8½", pp. 508, with three plates and some text figures. Price: 47s. 3d.

Intended to serve as a teaching aid in diagnosis and management of patients, to form a link between preclinical and clinical medicine, and thus to assist medical students, practitioners and technicians.

"Genetics and the Races of Man: An Introduction to Modern Physical Anthropology", by William C. Boyd, Ph.D.; 1950. Oxford: Blackwell Scientific Publications. 8" × 5½", pp. 470, with 52 figures. Price: 30s.

As the science of heredity became known a revolution took place in the ranks of physical anthropology. It is the purpose of this book to explain the revolution in anthropological thought, the scientific basis on which it rests and the way in which genetic knowledge can be applied to a study of mankind.

"Textbook for Health Visitors", by Llewelyn Roberts, M.D., M.R.C.P., D.P.H., I. G. Davies, M.D., M.R.C.P., D.P.H., and Beryl D. Corner, M.D., M.R.C.P.; 1951. London: Baillière, Tindall and Cox. 8" × 5½", pp. 558, with seven plates and 38 figures. Price: 21s.

The health visitor in Great Britain is a social worker with the same relationship to the general practitioner as the almoner has to the consultant in hospital. This book is primarily for health visitors, but also intended to help medical officers of health, district nurses, domiciliary midwives and welfare workers.

The Medical Journal of Australia

SATURDAY, JUNE 16, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE WORLD MEDICAL ASSOCIATION.

THE fourth general assembly of the World Medical Association was held in New York City on October 16-20, 1950. The report of the meeting is published in the *World Medical Association Bulletin* for January, 1951. This journal, which is published in three languages, English, French and Spanish, has as its Editor-in-Chief Dr. Morris Fishbein, of Chicago, and collaborating editors come from Copenhagen, London, Basle, Amsterdam, Montreal, Barcelona, Shanghai and Sydney. The meeting in October, 1950, was attended by 29 of the 39 constituent nations; the Federal Council of the British Medical Association in Australia sent its representatives. Dr. Elmer L. Henderson, of the United States of America, was installed as president, and the president for 1951 is to be Dr. Dag Knutson, of Stockholm, where the fifth general assembly will be held this year. One of the first acts of the fourth general assembly was to elect to membership the medical associations of Ethiopia and Thailand. The Council of the Association was also given authority to elect as members the medical associations of Japan and Western Germany, if it was deemed advisable to do so after correspondence with the membership associations. In the introductory part of the report of the meeting it is stated that the nations living behind the Iron Curtain sit apart, depriving themselves of the great advantages that come from association with colleagues. This is a great misfortune to the whole world, for we must recognize that frank discussions on science, art and general culture are the most powerful means for creating understanding amongst the nations. Prominent among the subjects discussed by the general assembly was a report of the committee on medical education. Certain recommendations were made and we read that an international conference on medical education is contemplated. Another decision which will receive approval is the determination to hold a conference with the World Health Organization. The Director-General of the World Health Organization, Dr. Brock Chisholm, was present in person and extended an invitation to the World Medical Association to discuss with his organization mutual problems. The invitation was accepted and a conference was to have taken place in May, 1951. It should be pointed out once more that the World Health Organization is an organization of governmental bodies. The World Medical Association, on the other hand, is formed by the medical associations of different countries. This is one of the reasons why this journal has always insisted that Australia should take a lively interest in the affairs of the Association. It would be foolish of the practising members of the medical profession to leave to a body representative of governments the world-wide aspects of medical problems. There is no doubt that conferences between the World Health Organization and the World Medical Association will be useful. One of the subjects which has engaged the attention of the World Medical Association for some time is social security; a frank exchange of views on this subject between both bodies is most desirable.

The general assembly of the World Medical Association was followed by a meeting of the medical editors of the world. Dr. Morris Fishbein presided and papers were presented by several editors and by a medical librarian. Among these was a paper on medical journalism by Sanford V. Larkey, director and librarian of the Welch Medical Library of the Johns Hopkins University. At the outset of his remarks Larkey expressed views which have been put forward from time to time in this journal and which cannot be too strongly emphasized. He pointed out that the publication of medical journals constituted a vital link in the whole research effort. He said that this was not always appreciated and that the financial responsibilities were often overlooked. He added that scientists at last were beginning to realize that the support of scientific publications had to be related to the overall cost of research. This is undoubtedly true, but it may be questioned whether its truth is also realized by those who provide and control funds for research. Discoveries in the research laboratory are useless unless they are made known to other research workers and to those who can apply the findings in a practical manner. Research in any one subject is seldom the work of one man or even of one group of workers in a laboratory. What happens is that one scrap of information is applied here, another there, and a third somewhere else; the scraps of information are assembled often in different parts of the world at the same time and an edifice is slowly erected. Sometimes the man who hews and places in position the coping stone of the building receives most of the credit. Whether this is fair or just need not concern us at present; what does concern us is that full and detailed information on research and on its methods should be made available to all who can use them. Larkey discussed medical periodicals from three points of view: first the sponsorship of medical periodicals, second the author's point of view, and third the point of view of the reader. He said that in his library a study had recently been made of 800 journals. Of these 590 carried a statement that they were sponsored by some society. The percentage of sponsored journals was 74; 210, or 26%, gave no indication of any society affiliations, but possibly this did exist in some instances. He pointed out that the percentage of sponsored journals in the United States and in Great Britain was somewhat similar. Of 397 journals in the United States 317 were sponsored, or 80%; in Great Britain, of 95 journals, 71 were sponsored, or 76%. This

sponsorship of scientific journals is becoming a matter of some importance at the present time. The costs of printing and publishing are increasing at a great rate and some scientific bodies are finding it very difficult to meet the costs of publication. The journals of bodies like the American Medical Association in the United States and the British Medical Association in Great Britain have such large circulations that rising costs can probably be met. The smaller societies representative of different specialties in medicine produce journals of the greatest value to medical workers in all places. Some of these societies are hard put to it at the present time to make both ends meet. A general solution of this problem will be hard to find, but there is no doubt that in certain instances journals with restricted circulation which publish results of research will have to look for assistance to the funds which in the first instance have enabled the research to be carried on.

The author's point of view in medical journalism is best known to authors and to editors. An author desires to have prompt and full publication, and in many instances, as Larkey declares, this is most desirable. It is the editor who faces the dilemma. On the one hand he has the author with his laudable desire for publication, and on the other hand he has the physical limitations of printing and publishing. An editor's most difficult task is to know when to say: "No." No author likes to cut down the size of his article. Still it sometimes has to be done, and it is surprising to what extent excisions can be made without any lowering of the value of the contribution. As a matter of fact if the pruning is done with care an improvement often results.

Larkey describes the reader as the ultimate consumer and states that he, probably more than anyone, is overwhelmed by the mass of literature. The reader wants information for all sorts of purposes and from all kinds of places. Sometimes, Larkey points out, the reader is just trying to keep up with what is going on. Sometimes he is in search of a specific fact and sometimes he wants to bring together all the knowledge he can find on a certain subject in relation to his own research. Here the trouble starts all over again in a sort of chain fashion. The reader will now write an article and want it published. Larkey holds that in any of these researches the method is apt to be too haphazard—there would seem to be almost a state of induced panic at the sight of the tremendous array of journals or of the pages and pages of seemingly complex index entries. Larkey reproduces the following interesting quotation.

> What is to be the result of this steadily increasing production of books? What will the libraries and catalogues and bibliographies of a thousand, or even of a hundred, years hence be like, if we are thus to go in the ratio of geometric progression, which has governed the press for the last few decades? The mathematical formula which would express this, based on the data of the past century, gives an absurd and impossible conclusion, for it shows that if we go on as we have been going there is coming a time when our libraries will become large cities, and when it will require the services of everyone in the world, not engaged in writing, to catalogue and care for the annual product. The truth is, however, that the ratio has changed, and that the rate of increase is becoming smaller. In western Europe, which is now the great centre of library production, it does not seem probable that the number of writers or readers will materially increase in the future, and it is in America, Russia and southern Asia, that the greatest difference will be found between the present amount of annual literary product and that of a century hence.

Larkey must have startled his audience by pointing out that the passage he had quoted was not his own, but had been written in the year 1881. It was written by John Shaw Billings, the man who started the *Index Medicus* and the *Index Catalogue of the Surgeon General's Library*. Larkey referred to the scope of present-day medical publications and said that any figures he could give would be only approximations. He said that there were probably between 8000 and 10,000 journals devoted to medicine and the related sciences. A few years earlier *The Index Catalogue* had listed almost 7000 periodicals and serial publications. Larkey thought a conservative estimate would be that something like 200,000 articles were published annually in these journals. It used to be said some years ago by a prominent Australian surgeon that probably two-thirds of all the articles published in medical journals need not be printed. From some points of view this would probably be correct. It is, however, necessary to consider where a journal is published and what circle of readers it serves. Members of scientific societies like to know that the proceedings of their body are recorded somewhere—the place of recording need not be in a journal which is the property of the society. This view is justified when we remember that any original observation, however unimportant it may appear to be, may, when linked to some other seemingly unimportant observation, prove of value to somebody else who is working on the subject. Medical journals, of course, have other functions; in editorial and correspondence columns they give information to and sometimes instruct their readers and galvanize them into action. In other words, if on the one hand they bring knowledge that will relieve suffering, on the other hand they act as stimuli to the somnolent. And this reminds us of the old definition of a newspaper's mission—to comfort the afflicted and to afflict the comfortable.

Current Comment.

VARICOSE ULCERS.

THE term "varicose ulcers" implies that these ulcers are always the result of, or at any rate are always associated with, varicose veins; otherwise the term is meaningless. Whether this connexion does or does not exist has long been the subject of dispute. The general more or less unthinking assumption is that these ulcers are indeed "varicose", and this view has its deliberate champions. Others have minimized, if not entirely dismissed, the role of varicose veins and have suggested that a term such as "gravitational ulcers" should be adopted. The practical position was well summed up by C. H. Wickham Lawes[1] in a paper read last December at a meeting of the New South Wales Branch of the British Medical Association:

> It is said that varicose veins occur without any ulcer or dermatitis and that an ulcer and dermatitis can occur without varicose veins. These are true statements and are quoted by many as being evidence that there is no connexion between the two conditions. A third fact is overlooked—that is, that in a vast number of cases ulcer and dermatitis heal very quickly and stay healed when the varicose veins are cured.

Many experienced clinicians will support Lawes's statement, though some will hesitate to echo the reference to "a vast number of cases". H. E. Lockhart-Mummery and J. Hillyer Smitham[2] incline in the opposite direction,

[1] THE MEDICAL JOURNAL OF AUSTRALIA, May 5, 1951.
[2] *The British Journal of Surgery*, January, 1951.

though with qualification. Their view is that visible varicose veins are not the sole cause of ulcers of the "varicose" type, if indeed they are the cause at all, and that other factors must be sought. They review the opinion put forward by a number of investigators that the most important factor in the production of ulcers is previous deep vein thrombosis, and they discuss relevant findings from venographic investigation. There is good evidence to relate ulcer formation with previous thrombosis, and others have already published venographic studies supporting this. However, the venography practised by Lockhart-Mummery and Smitham is of particular interest, because they have departed from the standard technique, in which the patient lies flat on the X-ray table, and have obtained venograms with the patient as nearly as possible in the erect position; the radioopaque material was injected into the femoral vein in the groin, allowing production of a "descending" venogram, in contrast with the customary "ascending" venogram obtained from injection into a vein in the foot. The venograms, which are splendidly reproduced, are of much interest. Lockhart-Mummery and Smitham admit that the correct interpretation of venograms is "notoriously difficult", but they feel justified in drawing certain conclusions. The first is that after thrombosis of the deep veins of the leg recanalization (with destruction of valves) does occur in the majority of patients. This, of course, corroborates the work of earlier investigators, but Lockhart-Mummery and Smitham point out that venography by ordinary methods is inaccurate and misleading on this point. It seems likely, they state, that recanalization proceeds from above downwards, probably by the formation of small channels that later enlarge and coalesce, and that not until a channel has formed throughout the whole length of the thrombosed vein can that channel be seen by ordinary venographic methods. Recanalization is apparently a slow process; the time required for completion varies, but is probably at least twenty years. Lockhart-Mummery and Smitham believe, and they appear to be justified in their belief, that the occurrence of previous thrombosis cannot be excluded by the patient's history or by a venogram prepared by the ordinary technique. In general, the development of intractable ulceration and serious symptoms after thrombosis is delayed until recanalization is far advanced. In particular cases in which this is not true, other factors play a part, notably the inadequacy of the collateral circulation and vasospasm. However, the more severe sequelæ seem to depend on complete recanalization. The resultant destruction of the valves or their damage so as to make them functionally useless is of great importance as it means, in effect, that with completion of recanalization there is an unbroken column of blood unsupported by valves from the right auricle to the lower part of the leg when the patient stands up. The venous pressure in the leg will thus be abnormally high and stasis severe—conditions sufficient to account for bursting pain, œdema, induration and ulceration. In certain cases in which previous thrombosis has apparently not occurred, and yet induration and ulceration develop, similar femoral vein insufficiency with back-pressure can be demonstrated and is thought to be the causative factor. The cause of the valvular incompetence is not known; it may be congenital or the result of degenerative changes. The last point made by Lockhart-Mummery and Smitham from their observations is that patients presenting with varicose veins whose skin on the lower part of the leg is healthy have a normal or nearly normal femoral vein. It seems likely, they state, that saphenous incompetence with obvious varicose veins may occur with or without associated deep vein incompetence; it is the latter factor which determines the venous stasis in the lower part of the leg and predisposes to ulceration, and the high pressure in the deep veins may well be the factor causing the recurrence of varicosities not infrequently seen after adequate saphenous ligation.

If these conclusions are valid, ligation of the deep vein should be beneficial for patients with ulceration and the other changes that go with it. Lockhart-Mummery and Smitham have had no experience of this operation, though they quote the favourable results claimed by others from both femoral vein and popliteal vein ligation. They rightly point to the fact that for the majority of leg ulcers treatment by bandaging gives satisfactory results. Only when this convenient and simple treatment has failed should more heroic measures be considered. Lumbar sympathectomy is stated to have produced good results in the presence of vasospasm. If ligation of deep veins is to be undertaken, Lockhart-Mummery and Smitham put forward reasons to favour popliteal vein ligation, but ultimately this major procedure can be justified only by results. Results so far reported (the work of Bauer is quoted in particular) appear to be encouraging, but they need wider corroboration. Indeed, the whole subject needs more attention. Lockhart-Mummery and Smitham fully realize that their series is a small one from which to make deductions. They believe their findings to be valid and in agreement with those of Bauer from entirely independent work, but they can see the aspects of the subject requiring further study. Their desire to stimulate the interest of others justifies their presentation of findings based on limited data; it is to be hoped that at least in this they will succeed, for the clinical implications of the work are too important to be treated lightly.

THE LOWER NEPHRON SYNDROME IN CLOSTRIDIAL INFECTION.

DURING the 1939-1945 war considerable attention was paid to the striking phenomena associated with what was called the "crush syndrome". The delayed shock which was observed after injuries causing crushing of muscular parts was carefully studied in England particularly; unfortunately the damage caused by air raids gave opportunity for considerable investigation, but the knowledge gained was not only valuable to the sufferers from these injuries, but furnished another chapter in the story of renal function and its disturbance by many different conditions, of which crush injuries formed one. Gas gangrene was, happily, not a very common result of infection of war wounds, but the serious nature of the systemic symptoms in severe cases calls for the greatest respect when these occur. Of these symptoms a shock syndrome may be part, and numbers of efforts to elucidate the action of the clostridial toxin have been made by use of the experimental method. M. Berg, S. A. Levinson and K. J. Wang have carried out some useful experimental work on the action of the toxin of *Clostridium perfringens*, which is known to produce a condition of shock with hæmoconcentration and the associated phenomena.[1] In this work these authors used dogs anæsthetized by intravenously administered pentobarbital sodium. It was found that a dose of eight millilitres per kilogram of body weight would kill the animals in five and a half to six hours, and this dose was injected intramuscularly in all the experiments. Control injections of the culture medium were also used without any toxin; these were without effect. Observations were made of the blood pressure of the animals, taken direct from the femoral artery, hæmatocrit readings were made at regular intervals, and studies of the chemistry of the blood and the urine were also carried out. The toxin was injected into the muscles of a leg, and in view of the local œdema produced in the limb the volume of this limb was estimated at intervals. Decrease in the urinary output began in the first hour after the injection, and after four hours all secretion of urine had ceased. Meanwhile the blood was showing an increasing concentration, which reached its height in four hours. The volume of the leg also showed alteration, and steadily increased up to a maximum of 1·6% to 3·0% of the body weight. Studies of the urine showed a decline in chloride excretion, and simultaneously the blood urea level rose significantly after four hours. A series of animals was also studied with special reference to the changes in the histology of the kidney. The animals were killed, some one hour and some half an hour after the injection of the

[1] *Archives of Pathology*, February, 1951.

toxin, a few minutes after the injection of phenol red and sodium ferro-cyanide, substances which are excreted chiefly by the proximal convoluted tubules and the glomeruli respectively. In a further set of experiments a cast of plaster was applied to the injected limb immediately after the injection of the lethal dose of toxin. In a final series the toxin-treated leg was massaged after the injection so as to diffuse the toxin more rapidly. These two sets of animals were submitted to the same investigations, and integration of all the experimental data was then made so as to assess the connexion between the œdema of the limb, the hæmoconcentration, the renal function, and the demonstrable damage to the various cellular components of the kidney. The authors found that the amount of local œdema could explain the larger part of the hæmoconcentration and the fall in the chloride content of the blood. Extrarenal factors and diminution of renal function may then be directly related to the findings in the blood. They consider that the rapid œdema of the limb may be responsible for the fall in urinary excretion, as no such change was observed in those animals which had a plaster cast applied to the limb. The kidneys showed no cellular changes demonstrable by histological methods until one and a half to three hours had elapsed after the giving of the toxin. Then the glomeruli showed vascular changes and swelling of the tufts, and later the swelling became sufficient to lessen filtration. After four or five hours pronounced circulatory failure was evident in the kidney, and the tubules as well as the glomeruli showed failure to secrete dye, with severe mitochondrial damage in the cells. The authors discuss details of the mechanism of these changes, but it is sufficient here to point out that they produced local œdema in the animals, with hæmoconcentration, a drop in blood pressure reaching shock level, and a significant reduction in renal function, which is apparently due to circulatory changes, direct action of toxin or both. Massage of the leg to diffuse the toxin enhanced its effects, whereas concentric pressure with plaster reduced, delayed and possibly modified the toxic effects. One interesting finding was the appearance of severe and extensive destruction of the kidneys after massage of the limb. These experiments serve to emphasize the method of action of the toxin of *Clostridium perfringens*, and to bring into line the symptoms with those observed in several well-known conditions.

EXPERIMENTAL STUDIES OF ASBESTOSIS.

FOR over twenty years a series of studies of asbestosis has been going on at the Saranac Laboratory in New York, being particularly associated with the name of Leroy U. Gardner. Partial reports of this work have been published, but now for the first time a complete survey has been presented of the entire experimental investigation, the authors being A. J. Vorwald (whose work in this field is widely known), T. M. Durkan and P. C. Pratt.[1] The paper is devoted to experimental asbestosis, and in it are described the experiments made on animals with various kinds of asbestos dust. Another report, to be prepared and issued at a later date, will be concerned with human asbestosis and will cover the health aspects of workers who have been exposed to asbestos dust in an industrial environment. Asbestosis is defined in this report as a form of pneumonokoniosis resulting from prolonged inhalation of asbestos dust. The name "asbestos" (literally "unburnable"), it is stated, is not that of a specific material, but is a term applied to a number of different minerals whose characteristic feature is a structure composed of long, parallel, flexible fibres. This structure is unique because the fibres are capable of repeated longitudinal subdivision to units of molecular proportions. In length the fibres vary from a few microns to six or more inches. The asbestos minerals are silicates of variable composition. Most of the asbestos of commerce is chrysotile ($3MgO.2SiO_2.2H_2O$), which occurs as veins in serpentine, a mineral of similar chemical composition. Other varieties used commercially, but in much smaller amounts, are crocidolite and amosite. Asbestosis in man, Vorwald, Durkan and Pratt point out, is a chronic disease with diffuse pulmonary fibrosis which requires years to develop; but it is possible to reproduce in one or more species of animal characteristic tissue changes which are similar to the lesions of human asbestosis. Since the life span of the experimental animal is relatively short, it is not possible to produce the characteristic lesions in animals under conditions identical with the usual industrial environment. Consequently, to obtain a complete evaluation of the tissue response to inhaled particulate and fibrous material, it is necessary to accelerate the reaction by using higher concentrations of dust than would ordinarily be encountered in industry. While conditions of exposure are thus different, the information yielded by animal experiments is regarded as invaluable in furnishing a better understanding of the reaction of the human organism to inhaled asbestos dust. The experiments carried out are described in detail in the paper, but it will be sufficient here if we refer to the main findings and conclusions drawn. It was found from both inhalation and injection experiments that various species of animal, including the guinea-pig, the rat and the rabbit, but not the mouse and the dog, develop peribronchiolar fibrosis of the lung similar to human asbestosis after being exposed to long chrysotile asbestos fibres. It appears that long asbestos fibres are essential in the production of the peribronchiolar fibrosis; short fibres are incapable of producing this reaction. The minimum length of fibre possessing the capacity to produce the typical reaction in animals is somewhere between 20μ and 50μ. The mode of action of the long asbestos fibre in the production of asbestosis appears to be primarily mechanical rather than chemical in nature. This is a finding of interest and importance as the chemical hypothesis has had a considerable following in preference to the mechanical hypothesis. In the chemical theory, which is based on experience with quartz, it is assumed that the asbestos minerals dissolve in the body fluids and that in this process their bases are leached away to leave silica in a form capable of irritating tissues; asbestosis is thus regarded as merely an indirect form of silicosis. Vorwald, Durkan and Pratt bring forward a considerable body of evidence to show that the chemical theory is untenable and to support the importance of mechanical irritation. In an investigation of significant atmospheric concentrations it was found that typical experimental asbestosis was produced by the inhalation of an atmospheric suspension containing an average of 138,000,000 asbestos particles per cubic foot of air by light field count, of which less than 1% consisted of fibres longer than 10μ. The evidence shows at least that an atmospheric concentration of asbestos dust containing less than 1,000,000 fibres longer than 10μ per cubic foot of air is capable of producing experimental asbestosis in guinea-pigs; but it is not considered that the actual lower limit of concentration of long fibres necessary to produce asbestosis in animals can be established from these studies. The effective duration of exposure is important even though these animal experiments involve a different time factor from that under conditions of industrial exposure of humans. It was found that the duration of exposure required to develop the pulmonary reaction to inhaled asbestos dust was inversely proportional to the concentration of long fibres in the atmosphere; as the concentration was increased, the reaction developed in shorter time. Another finding of interest was that established experimental asbestosis ceased to progress on discontinuance of dust exposure. Indeed, there was an appreciable clearing of the mature pulmonary lesions, due to contraction of the fibrous tissue. An immature tissue response, evidenced primarily by cells with little or no fibrosis, continued to progress. Asbestosis bodies have always been a feature of interest in the study of asbestosis. From these experiments it is concluded that the formation of asbestosis bodies represents a coating of the fibres by blood and tissue elements, and this results in loss of ability of the

[1] *A.M.A. Archives of Industrial Hygiene and Occupational Medicine*, January, 1951.

fibre to produce fibrosis. Intratracheal injection of asbestosis bodies failed to produce the typical asbestotic tissue reaction in animals. It is suggested that the cessation of progressive reaction observed soon after exposure terminates may be due to the formation of asbestosis bodies. Claims regarding the protective value of aluminium received no support from these studies. Aluminium hydroxide added to the suspension of chrysotile asbestos before intracheal injection did not retard or prevent the development of asbestosis in rats. The last point relates to the association of tuberculosis and asbestosis. It was found that inhalation of asbestos dust did not alter significantly the final outcome of experimental tuberculosis in two series of guinea-pigs exposed to the dust. The apparently mild influence of asbestos dust, it is pointed out, is in distinct contrast to the stimulating effect exerted by inhaled quartz on a tuberculous process in the lung. Interpretation of the experimental findings is as yet tentative, but so far they suggest that inhaled asbestos dust has no more than a mildly unfavourable effect on pulmonary tuberculosis. The information and comments on this as well as other aspects to be brought forward in the promised report on human asbestosis will be awaited with much interest.

CITROVORUM FACTOR.

THE interest of hæmatologists has been aroused by the isolation from liver of a factor that is apparently quite distinct from folic acid and vitamin B_{12}. This is the citrovorum factor, so called by its discoverers, H. E. Sauberlich and C. A. Baumann, from their observation of its growth-promoting factors in relation to the saprophytic bacterium *Leuconostoc citrovorum*.[1] They were able subsequently to prepare a concentrate of the factor and to demonstrate that it was distinct from vitamin B_{12} and folic acid,[2] though its action was closely linked with that of folic acid, and Sauberlich reported greatly increased excretion of citrovorum factor in the urine of rats and of a human subject as the result of ingestion of folic acid.

It is natural that the association of this factor with other potent liver factors should stimulate investigation of its possible therapeutic actions, and two reports have now appeared, one describing reversal of the toxic effects of folic-acid antagonists, the other its effect in the treatment of pernicious anæmia. E. B. Schoenbach, E. M. Greenspan and J. Colsky[3] encountered toxic effects in the usual form of ulceration of the mouth and leucopenia while treating two patients with the folic-acid antagonists aminopterin and amethopterin; both patients had metastasizing neoplasms. In both cases the administration of citrovorum factor without discontinuance of the folic-acid antagonists brought about rapid healing of the ulcers and disappearance of the leucopenia—a response that has not been previously effected with folic acid, vitamin B_{12} or liver extract. The toxic effects recurred ten days after cessation of administration of citrovorum factor. It is true that not a great deal of benefit was obtained from the folic-acid antagonists in these cases, and that in general their therapeutic usefulness is yet to be established; nevertheless, if the citrovorum factor will effectively counter their toxic effects, they can at least be submitted to adequate trial.

The first report on the use of citrovorum factor in the treatment of megaloblastic anæmia comes from the department of medicine in the University of Edinburgh. In a preliminary communication L. S. P. Davidson and R. H. Girdwood[4] describe their treatment with citrovorum factor of three patients suffering from Addisonian pernicious anæmia. In each case the dietary history was good, there was no diarrhœa, test-meal examination revealed histamine-fast achlorhydria, and the marrow was megaloblastic. After a control period, during which there was no evidence of remission, the patients were given, respectively, 20, 40 and 80 million units of citrovorum factor in a single intramuscular injection, and the hæmatological response was followed for two weeks. A slight hæmatological response occurred in the first two cases, but only with the dosage of 80 million units was a satisfactory response obtained; it was accompanied by a corresponding improvement in the state of well-being. Davidson and Girdwood point out that these results suggest that, provided a large enough dose is given, the citrovorum factor will produce a hæmatological and clinical response in patients with pernicious anæmia. However, consideration of the therapeutic doses of folic acid and citrovorum factor, which appear to be of the same order, leads them to conclude that the activity of refined liver extract in the treatment of pernicious anæmia is related not to its content of either of these substances, but to its content of vitamin B_{12}. They quote the recently reported work of Nichol and Welch which suggests that the citrovorum factor may be the derivative of pteroylglutamic (folic) acid responsible for the continuation of normoblastic blood formation. It is early yet to draw definite conclusions, but it appears likely that further investigation of citrovorum factor and its mode of activity will aid in the understanding of megaloblastic anæmia and perhaps of the related therapy.

[1] *The Journal of Biological Chemistry*, October, 1948.
[2] *Ibidem*, December, 1949.
[3] *The Journal of the American Medical Association*, December 30, 1950.
[4] *The Lancet*, March 31, 1951.

ADHESIVE TAPE DERMATITIS.

ADHESIVE TAPE DERMATITIS is common enough, but comparatively little thought has been given to the mechanism of its production. It has indeed been generally taken for granted that simple local irritation of mechanical and chemical origin is responsible. S. M. Peck, T. J. Michelfelder and L. J. Palitz[1], who have for some years now been taking an interest in the subject, hold that adhesive tape irritation can be divided into a number of types. Allergic contact dermatitis may be produced, with characteristic clinical and microscopic features, but this reaction is rare. Moreover, it is practically impossible to trace the specific sensitizer in any particular adhesive because the chemical composition of the ingredients varies from batch to batch. Another type of effect is "a fleeting unimportant reaction", which has been called the reaction of removal. This is due to the mechanical removal of the tape, especially when a "tacky" adhesive has been used, and can be decreased to a certain extent by the use of adhesive remover. However, according to Peck, Michelfelder and Palitz, the majority of cases of adhesive tape dermatitis are due to a specific mechanism which can be summarized as follows. The initiating factor in most instances, if not in all, is a plugging of the ostia of the sweat ducts by keratin. The development of the sweat retention syndrome and the production of what might be referred to as *miliaria rubra* follow. Everything else being equal, the degree of irritation developing is dependent on the number of sweat ducts involved and the extent of the plugging, on the activity of the sweat glands, and probably on the relative irritating properties of the ingredients of sweat. The various adhesive tapes, it is stated, contain ingredients which tend to stimulate hyperkeratosis to a greater or lesser degree. Hyperkeratosis of this type is not limited to the ostia of the sweat ducts, but is diffuse; even follicular hyperkeratosis may be produced. From this it follows that, according to the formulation of the materials used and their capacity to stimulate hyperkeratosis, some adhesive tapes may tend to be more irritating than others. The suggestion is made that it might be worth while to investigate each individual ingredient of adhesive tapes from the standpoint of special tendency to produce hyperkeratosis. It may be also that once plugging of the ducts takes place, bacteria multiply under the tape and add to the maceration and irritation. However, the role of bacteria in the production of this dermatitis is not established.

[1] *A.M.A. Archives of Dermatology and Syphilology*, March, 1951.

Abstracts from Medical Literature.

RADIOLOGY.

Accessory Sacro-Iliac Articulations with Arthritic Changes.

LEE A. HADLEY (*Radiology*, September, 1950) states that accessory sacro-iliac articulations are frequently visualized between the ilium and the posterior surface of the sacrum. These joints may show arthritic changes of greater or lesser degree. Ankylosis may occur. Many of the patients complain of low back pain, and some complain that pressure over the accessory joint is painful.

Vitamin D Resistant Rickets (Refractory Rickets).

JOHN F. HOLT (*American Journal of Roentgenology*, October, 1950) states that vitamin *D* resistant rickets, or refractory rickets, as it is more commonly called, is an apparently rare metabolic disorder of childhood, which differs from ordinary infantile rickets in that spectacularly large doses of vitamin *D* (50,000 to 1,000,000 units per day) are required to effect a cure and to prevent recurrences. The blood chemistry in patients with refractory rickets is almost identical with that found in infantile rickets, namely, normal serum calcium content, low serum phosphorus content and elevated serum phosphatase content. One difference lies in the fact that the serum phosphorus level remains persistently low despite intensive treatment. Furthermore, decreased urinary calcium excretion is not as prominent a feature as it is in ordinary rickets. Radiologically, refractory rickets has much the same appearance as other types of rickets. One sees generalized rarefaction and coarsening of the trabecular pattern of various bones, irregular fraying of the epiphyseal plates, widening of the spaces between the epiphyseal ossification centres and the shafts, and cupping of the ends of the shafts. Secondary deformities, such as bow leg, *coxa vara*, knock-knee and sabre shin, are more common and more profound in refractory rickets than in infantile rickets, as are greenstick fractures, pseudofractures and angular deformities of the upper extremities. In the refractory variety, one is apt to observe rhachitic changes in unusual locations, such as the angles of the scapulæ, the elbows and the ischio-pubic synchondroses. The humeri may appear short and broad, as in chondrodystrophy, and the thoracic cage is more apt to be deformed. The *lamina dura* around the teeth is almost invariably absent or poorly defined. Radiologists should be thoroughly familiar with refractory rickets because, in a given case, it is usually on the basis of unequivocal radiographic signs that rickets is first considered as the diagnosis. The physician who is unaware of a refractory form of the disease is apt to disregard the radiological diagnosis of rickets in the face of a history of apparently adequate vitamin *D* intake or failure of the patient to respond to moderately large doses of the vitamin. By the same token, the unwary radiologist may alter his initial diagnosis of rickets in the face of this seemingly overwhelming clinical evidence against it. Consequently, the patient may be subjected to ill-fated surgical procedures for correction of bowing and other deformities at a time when his rhachitic process is still active. Once the diagnosis of refractory rickets is established and the patient is started on the tremendous doses of vitamin *D* necessary to effect a cure, careful supervision of the therapy is essential to prevent the development of vitamin *D* intoxication.

Pirie's Bone.

CHARLES GOTTLIEB AND S. L. BERANBAUM (*Radiology*, September, 1950) state that there is a general misconception regarding the significance of a small accessory ossicle located on the dorsum of the astragalus, just proximal to the astragalo-scaphoid joint. A skiagram taken after a traumatic incident about the ankle joint will reveal this ossicle, at times with a somewhat irregular surface, adjacent to the main body of the astragalus. This repeatedly occurs in compensation cases with resultant misdiagnosis of a chip fracture. A. H. Pirie, in 1921, first focused attention on this ossicle, which now bears his name. At that time he had collected 14 cases, in four of which the condition was bilateral. Proof of the existence of the bone was obtained *post mortem*. The distal end of the astragalus often exhibits developmental irregularities. The astragalo-scaphoid ossicle is a frequent occurrence and causes no difficulties. The irregularity in contour in the form of an exostosis on the dorsum of the neck, called the astragaloid spur, is less well known. At times this spur may be present as a distinct ossicle with cortex on each surface and no demonstrable defect in the astragalus, in spite of the irregularity in appearance periodically observed on the surface adjacent to the main body of the tarsus. It has been shown that pathologically the line of demarcation from the talus is cartilage. This pitfall in diagnosis can often be avoided by taking the opposite ankle for comparison, since there is a distinct bilateral tendency. The fact that no essential change is demonstrable in the skiagram, after the repeated application of a cast for varying lengths of time, should establish the diagnosis of an accessory ossicle in contradistinction to a chip fracture.

"Priodax" and Pseudoalbuminuria.

E. E. SEEDORF, W. N. POWELL, R. G. GREENLEE AND J. T. HARTMAN (*Radiology*, November, 1950) state that a "false positive" reaction for albumin will occur in the analysis of the urine of a significant number of patients receiving the standard dosage of six tablets of "Priodax". The fact that it is the actual presence of "Priodax" in the urine which causes this reaction is proved by laboratory tests. The false albuminuria occurs with the greatest frequency in the lighter weight group, and therefore in women and younger individuals. Other factors, such as prevailing barometric and temperature readings and the specific gravity of the urine examined, play no apparent role. Detectable amounts of "Priodax" are present in the urine of certain patients, not only on the morning after the ingestion of "Priodax", but the following day and occasionally even on the third day. A report of albuminuria in patients who have received "Priodax" should therefore be interpreted with caution as far as the presence or absence of renal disease is concerned. To avoid confusion or misinterpretation, the urine of gall-bladder patients should be examined prior to the administration of "Priodax". If this is not possible or practical, it would seem advisable that for three days after ingestion of "Priodax" some record to the effect that "Priodax" has been administered should be made on urine examination request slips. "Albuminuria" occurring after administration of six tablets of "Priodax" does not indicate renal irritation from the drug.

Radiological Changes in Pulmonary Stenosis.

R. F. HEALEY, L. DEXTER, M. ELKIN AND M. C. SOSMAN (*American Journal of Roentgenology*, June, 1950) have made an analysis of the radiographic and hæmodynamic changes observed in eight cases of mild uncomplicated pulmonary stenosis and in one case of severe pulmonary stenosis complicated by a right-to-left shunt through a septal defect, but without an overriding aorta. They state that the most significant radiographic finding in six of these nine cases was a post-stenotic dilatation of the "main pulmonary artery" which did not extend to include the hilar or intrapulmonary branches. This finding has been observed by others. In the majority of cases cardiac or selective chamber enlargement was not found. Left auricular enlargement was not observed in any of the cases. A differential diagnosis is given of pulmonary stenosis based on the appearance of the pulmonary artery and the absence of left auricular enlargement; included are atrial septal defect, ventricular septal defect, patency of the *ductus arteriosus*, Eisenmenger's complex, mitral stenosis, *cor pulmonale*, the tetralogy of Fallot, and dilatation of the pulmonary artery from unknown cause.

PHYSICAL THERAPY.

Carcinoma of the Naso-Pharynx.

THEODORE P. EBERHARD AND ROBERT H. LEAMING (*Radiology*, July, 1950) state that treatment of carcinoma of the naso-pharynx by irradiation involves three problems, the localization of the tumour, the delivery of an adequate tumour dose and the maintenance of the patient's general health during treatment. About 40% of malignant naso-pharyngeal growths are squamous-cell carcinomata, 40% lymphosarcomata, 10% lymphoepitheliomata, and the remainder adenocarcinomata, plasmocytomata and rare growths. The authors maintain that in the planning of treatment, no distinction should be made between carcinoma and sarcoma as regards dosage, although knowledge of the histology is of great importance from the point of view of prognosis. Most of the successes occur in the lymphosarcoma and lymphoepithelioma group. As a general rule, an attempt is made to treat the primary tumour and the glandular areas at the one time, although the authors do not use large fields covering the whole area, but separate fields placed according to the areas involved—usually six

fields for the naso-pharynx and two lateral fields to the cervical glands. If it is thought that a sufficiently high dose has not been given to the primary tumour, supplementary radium treatment is given by a tube placed locally in the naso-pharynx. This is held in a cork or a Blady applicator. The general care of these patients is often difficult. The course of treatment is prolonged, and for many weeks they may not be able to take proper nourishment owing to the profound reaction and later dryness of the mouth with loss of taste. It is important to warn the patients beforehand of the severity of the ordeal, as in this way their cooperation is more easily gained. Of the first 20 patients treated, five remained alive and well after ten years. In a later series of nine cases, in which the same technique was used, only one has survived over three years. Bone destruction at the base of the skull is a very bad prognostic sign, but lymph node involvement is far less serious as at least 20% of those so affected appear to be cured. The authors state that success depends upon early diagnosis, accurate localization of the tumour and careful planning and carrying out of treatment.

X-Ray Therapy of Osteomyelitis of the Fingers.

A. H. BAKER AND F. FRIEND (*American Journal of Roentgenology and Radium Therapy*, November, 1950) state that osteomyelitis is a frequent and troublesome complication of a septic finger. Conservative treatment in general is recognized as being of the greatest value, but healing may take months. X-ray therapy in these cases has much to offer in easing pain and greatly shortening the period of healing. In acute and chronic inflammation, irradiation has an effect upon the mononuclear leucocytes leading to early differentiation of these cells to fibrocytes and fibrous tissue, thus allowing repair and preventing excessive local reaction. The technique of treatment is to give 50*r* to 75*r* to the affected area once a week for three to five weeks. In very acute infections the dose can be reduced to about 25*r*. Small doses are essential, and the results are better when treatment is early; but in at least three cases in which amputation appeared inevitable, the finger was saved.

Surgical and Radium Treatment of Antral Cancer.

VINCENT COLLINS (*Radiology*, July, 1950) discusses the treatment of antral cancer by combined surgery and radium therapy. He states that the first step is surgical—either peroral resection of the antral floor, with curettage of the antral and ethmoidal linings, or a radical maxillary resection through the Weber-Ferguson incision. A radium applicator can usually be tolerated 10 to 14 days after surgical operation. An impression of the area is taken in dental compound and an applicator in dental acrylic fabricated on the model. This is bored to hold the radium according to the surface dose planned. Usually the applicator is worn intermittently for five to seven days. The dosage in an average case is about 8000*r* at the periphery of the irradiated area. The reaction may take many weeks to subside. During this time the cavity is irrigated six times a day with normal saline, and slough and necrotic tissue are removed at frequent intervals. After four to six months, the operative defect can usually be closed by an upper plate prosthesis.

Irradiation Treatment of Cervical Lymph Node Metastases.

CHARLES L. MARTIN (*Radiology*, July, 1950) states that a review of the literature gives conflicting opinions on the value of irradiation for cervical lymph node metastases, but, in general, most radiotherapists are of the opinion that either X-ray therapy or radium implantation gives poor results. The author claims that a combined technique of radium needle implantation supplemented by external X irradiation has given gratifying results. The method is to implant the cervical area by a planar implant with low intensity radium needles to give a dose of between 6000*γr* and 12,000*γr* to the subcutaneous tissues. On the day after the implant is carried out, X-ray therapy is begun, a daily dose of 350*r* (in air) being given up to a total of 2100*r*. Moist desquamation occurs, but this heals in six to eight weeks. This technique is not advised after incomplete block dissection, and should be used only when glands are palpable and if the primary lesion appears to be eradicated. A series of 146 patients clinically diagnosed as having metastatic involvement of cervical glands have been thus treated, and of these 40 (27·3%) were alive and well at the end of five years. Many of these patients had an inoperable condition, and the author claims that even in advanced cases great palliation can be obtained.

MEDICINE.

Diet and Essential Hypertension.

D. M. WATKIN *et alii* (*The American Journal of Medicine*, October, 1950) studied the effects of being in hospital *per se* to provide a basis for evaluation of results of allegedly specific treatment for essential hypertension in a hospital environment. They record precise data to support the general impression that the response of individual patients with essential hypertension to treatment in hospital *per se* is so variable and unpredictable that a prolonged period of observation is a prerequisite to valid appraisal of the specific effects of therapy. They also report the results of a two-year controlled investigation of the Kempner rice regime in 50 patients with advanced essential hypertension. They state that the results corroborate in most essentials those of Kempner. The extremely low sodium content of the rice diet appears to be the most significant factor in reduction of raised blood pressure levels. The rice diet may be unnecessarily restrictive in regard to protein and perhaps in other respects. Rigid restriction of sodium appears to be an important principle in the dietary management of at least the small proportion of subjects with essential hypertension who require more than reassurance, weight reduction or mild sedation. Effective sustained application is, however, difficult and protracted use of the Kempner regimen imposes such hardship and causes such difficulty in control as to be virtually impracticable for general use. The authors consider justified further extensive exploration of sodium restriction in essential hypertension and suggest points to be clarified.

Brill's Disease.

E. S. MURRAY *et alii* (*The Journal of the American Medical Association*, April 8, 1950) discuss the diagnosis of Brill's disease. They state that this is a typhoid-like illness found in the north-eastern States of the United States of America, especially among immigrants; 90% of those affected have been Jews. Fourteen cases are reported. Typhus rickettsiæ were isolated in seven only. In four the onset was sudden, with chills, fever and severe headache. In others the onset was gradual, with the same symptoms, but in three the rash on the fourth or fifth day was the first sign. The rash consisted of macules or maculo-papules on the anterior part of the chest near the axillæ, and later on the trunk, arms and thighs, appearing on the seventh or eighth day. The rash disappeared only in convalescence. The temperature was high and continuous before treatment; the pulse rate was 70 to 94 per minute; other signs were variable. White cell counts were normal or slightly raised. Complement-fixation tests showed elevated titres. Weil-Felix tests were carried out and resulted in an agglutination of *Proteus* OX19 organisms in 10 patients with a titre of over 1 in 700 in most cases, so that the result was not considered positive by ordinary standards, which demand agglutination at 1 in 700 or more. The test was regarded as unreliable in this series. Patients in this series were found to infect body lice with typhus rickettsiæ, which suggests the possibility of epidemics of the disease.

Venous Occlusion and Arterial Blood Flow.

J. E. THOMPSON AND J. R. VANE (*The Lancet*, February 17, 1951) have studied the acute effects of venous occlusion on the rate of arterial inflow to the hind limbs of cats. These studies were carried out directly by perfusion and by serial radiography. The authors state that during venous occlusion the arterial inflow to the extremity decreases. After release of the occlusion there is commonly a transient increase in arterial flow above the pre-occlusion level, but this reactive hyperæmia does not compensate for the decreased flow during occlusion. The rate of flow following release is equal to or less than the rate preceding occlusion, never higher. Serial radiography showed a delay in the filling of the vessels on the occluded side and a considerable reduction in the number of visible small arterial vessels under the inflated cuff. It is concluded that there is no direct experimental evidence in man or in animals to substantiate the view that venous occlusion increases the arterial blood flow to an extremity.

Spontaneous Rupture of a Papillary Muscle.

H. SCHWARTZ AND F. R. CANELLI (*American Heart Journal*, September, 1950) suggest that the appearance of a loud mitral systolic murmur together with intractable pulmonary œdema in a person who has suffered infarction of the heart should make tenable a clinical diagnosis of rupture of a papillary muscle in the left ventricle.

Special Articles.

PERFORATION OF THE ILEUM AMONGST PRISONERS OF WAR IN KOREA.

THE following is an account of a large number of cases of perforated ileum which occurred amongst North Korean prisoners of war in a prisoner-of-war hospital in South Korea.

The ages of the patients varied from eighteen to thirty-seven years. The patients usually had a history of diarrhœa of varying severity for some days prior to the perforation. Most of the patients were in good condition and appeared reasonably well nourished, except for a small minority who were still showing the effects of undernourishment.

The usual story was the sudden onset of acute abdominal pain. The patients were rarely brought to the hospital in less than twelve hours after the onset of the pain; sometimes it was twenty-four hours and in a few cases forty-eight hours.

They all showed the typical clinical picture of a ruptured bowel, that is, acute distress, a tender, rigid, silent abdomen, and thoracic respiration. The X-ray picture showed air under the diaphragm. A blood count revealed that the number of erythrocytes and the hæmoglobin value were usually within normal limits; the number of leucocytes varied from normal to 20,000 per cubic millimetre, and when increased was characterized by neutrophilia; eosinophile cells were absent or nearly so, although 90% of North Koreans harbour *Ascaris lumbricoides*.

The perforation was always situated on the anterior mesenteric border of the terminal part of the ileum within two feet of the ileo-caecal junction, in nearly all cases single except for a few in which two or three were present. The diameter of the perforation was usually several millimetres.

The external appearance of the bowel was hyperæmic in the vicinity of the perforation, and the regional mesenteric lymph glands were enlarged. When the bowel was opened, the mucous membrane was found to be acutely inflamed for several inches on each side of the perforation, and occasionally several acute ulcers were present in the mucous membrane, which had not penetrated the muscle coat.

In all cases *Ascaris lumbricoides* was present in the small intestine, and often one was found free in the peritoneal cavity.

The peritoneal cavity always contained a large quantity of free fluid, rice grains often being present, and peritonitis was always pronounced.

The method of treatment adopted which was found to be the most successful consisted of resection of about six inches of ileum, repair being effected by an end-to-end anastomosis with a single-layer Halstead mattress suture of cotton. A rubber drain was left in the flank. Spinal anæsthesia was usually used.

A Levine tube was passed prior to operation; a pint of blood and a pint of glucose-saline solution were given intravenously during the operation.

The post-operative treatment consisted of continuous gastric suction, intravenous glucose-saline therapy, further blood if required, penicillin and streptomycin, the patient being gradually brought on to a diet of rice soup and then normal "chop chop", which consisted of rice and a little fish.

One hundred and twenty patients were operated on during January and February; there was a 10% mortality, chiefly due to the long lapse of time before these patients were brought to operation.

The pathology was not definitely determined. Paratyphoid A bacillus was grown from some of the sections, though the ulcer did not have the typical typhoid appearance. The *Ascaris lumbricoides* infestation was probably incidental, though on occasions ascaris does perforate the small intestine.

Owing to the lack of good pathological facilities, it was considered that the perforation was due to paratyphoid A associated with a previous dietetic deficiency.

One patient on whom I operated myself had a perforated Meckel's diverticulum.

I am indebted to the Director-General of Medical Services for permission to publish this article.

N. M. KATER,
Wing Commander, No. 77 (F) Squadron, Royal Australian Air Force, Korea.

LIGHTNING BURNS IN KOREA.

AT approximately 3.30 p.m. on April 11, 1951, an electrical storm struck a few miles north of the 38th Parallel in Central Korea where the Third Battalion of the Royal Australian Regiment was fighting. Two companies were spread out along a 3000 feet high mountain, which was suddenly struck by lightning. Three isolated men were knocked to the ground and lost consciousness for periods up to two minutes, when they awakened with acute headaches and stiff limbs. I examined them a short while afterwards when they showed little generally that was abnormal apart from mild shock, but their burns were interesting. These were spider-shaped, about two to four inches in diameter and immediately below some metal object on the outer clothing, which itself was unaffected. In two cases, metal buckles had caught the lightning and in the third two hand grenades hooked on the side. Onlookers stated that flashes came from these objects. All three men were standing near insulated signal wire, which was in use at the time, the speakers on each end being mildly burnt by the receivers.

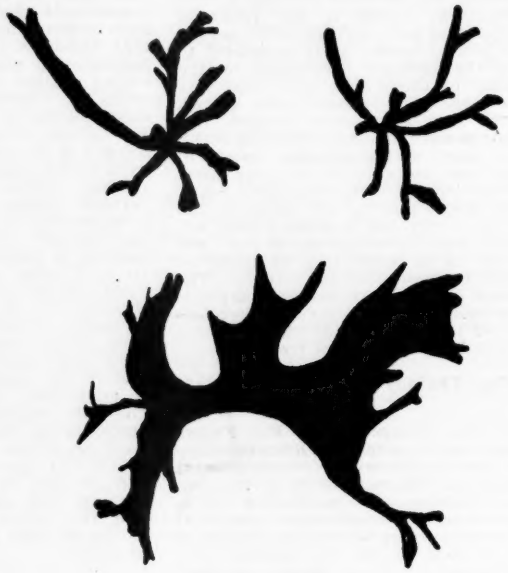

FIGURE I.
Case I. Private A.H. Above: Burns on left side of chest. Below: Burn on left shoulder.

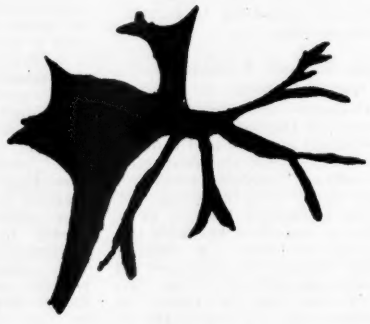

FIGURE II.
Case II. Private K.M. Burn on left side of chest.

The burns themselves were of first degree and sharply outlined. I evacuated the men to the 60th Indian Field Ambulance. I cannot remember having read of similar

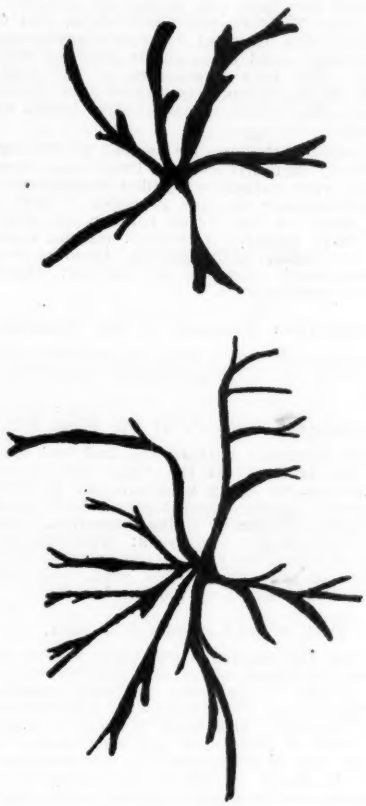

FIGURE III.

Case III. Private S.H. Above: Burn on left arm. Below: Burn on left side of chest.

shaped burns and in this isolated area have no literature to consult. I should like to hear of other cases.

Figures I, II and III show a rough outline of the burns at about half-scale.

D. D. BEARD,

Captain, Royal Australian Army Medical Corps, Regimental Medical Officer, 3 Battalion, Royal Australian Regiment, Korea.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Bendigo Base Hospital on April 28, 1951, DR. ROY WATSON, the President, in the chair for the medical section and DR. LEONARD BALL in the chair for the surgical section. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Subarachnoid Hæmorrhage.

DR. PHILLIP GOODMAN presented a fireman, aged thirty-one years, who had previously been in robust health. Some twelve weeks before the meeting, whilst scrubbing out the kitchen during a thunderstorm, the patient had suddenly became unconscious and started to have a series of Jacksonian epileptic seizures. These started with a cry, followed by head rolling and grimacing and then clonic movements of the left arm and leg. There were no signs of external injury and no evidence of electrical burn, and the patient was at once taken to hospital by ambulance. Lumbar puncture was then undertaken, and although difficulty was experienced because of the recurrent fits, a few millilitres of heavily blood-stained cerebro-spinal fluid, under what appeared to be slightly increased pressure, were finally withdrawn. The convulsion at once ceased and did not recur. A more detailed examination was then carried out. The left pupil was found to be larger than the right, the knee jerks were only just elicited, and plantar responses were absent. Examination of the ocular fundi revealed no abnormality. There was no head retraction, but slight neck stiffness was noted. Coma persisted for thirty-six hours, during which time the patient was given prophylactic penicillin therapy; enemata of magnesium sulphate were administered for the first four days. When the patient regained consciousness he complained of headache, which appeared to be relieved by aspirin, and convalescence was uneventful apart from a rather fluctuating blood pressure, which finally steadied down to a reading of 140 millimetres of mercury, systolic, and 80 millimetres, diastolic. He was then gradually allowed out of bed and finally had returned to work of a light character about two weeks before the meeting.

Dr. Goodman considered the diagnosis to be one of subarachnoid hæmorrhage, probably due to a rupture of an aneurysm of the circle of Willis. He believed that the condition was not so uncommon as was sometimes believed, as he had encountered four cases in the past three months. He stated that the prognosis must be guarded, because of the risk of recurrence, and heavy work or sudden strain should be avoided.

Congenital Heart Disease.

DR. J. S. GORMAN presented two children with congenital heart disease. The first of these was a girl, aged seven and a half years, who had failed to thrive since birth, although she maintained a good appetite. Her weight was only about that of a three-year-old child. Examination of the patient revealed a palpable continuous thrill and an extremely loud "machinery" type of continuous murmur maximal in the second left intercostal space; there was no evidence of cardiac enlargement. By way of contrast, the second child, also a girl, aged three years, was well up to the weight for her age and showed no disability. But a similar (although not so loud) continuous murmur was audible to the left of the sternum.

Dr. Gorman presented these two children as examples of patients with patent *ductus arteriosus*, and he pointed out that both the electrocardiogram and X-ray appearances of the heart were compatible with this diagnosis.

Sjögren's Syndrome.

DR. J. E. M. GRANT next presented two patients suffering from Sjögren's syndrome. The first, a woman, aged sixty-one years, had a sore and dry tongue, a dry mouth, inflamed eye, cracks at the angles of the mouth and a dry skin. A rheumatoid type of arthritis, confined to the hands, was also present.

The second patient, also a woman, had sought advice five years before, when she had a large left parotid swelling and complained of a cracked tongue, a dry mouth, and eyes which stung and were continually crusting over owing to the discharge of pus. She had no joint symptoms. Investigation of this woman at the Royal Melbourne Hospital had yielded no significant findings, and the diagnosis of Sjögren's syndrome was confirmed. It had been suggested that intramuscular injections of riboflavin might be tried, and that artificial tears be used to relieve the eyes—these to be made up of 1% gelatin in saline.

Dr. Grant went on to explain that Sjögren's syndrome had been first described in 1933. The main symptom (or sign), according to the original description, was dryness of the mucous membranes, and particularly of the conjunctiva even to the extent of complete absence of tears. Other associated signs and symptoms, as listed in the original and later descriptions, were diminished nasal secretion, disturbance of sense of smell, dry mouth, dry pharynx, larynx and trachea, absence of gastric enzymes, disturbed carbohydrate metabolism, lack of perspiration, muscular atrophy and weakness, and chronic polyarthritis. The disease was said to be mainly observed in women of post-climateric age. Nothing was really known of the ætiology of the condition, although endocrine dysfunction, vitamin deficiency or disturbance of the blood-forming tissues had been suggested.

Cardiac Failure.

DR. L. FARRAR presented a man, aged thirty-eight years, who, apart from a skin rash while he was in the army, had had no previous illness. He had been in the habit of taking up to twelve "pots" of beer a day for some years. He gave the story of having developed epigastric pain occurring after meals some four months previously; this was of varying severity and duration. He became disinclined for food and began to lose weight. A few weeks later he stopped work and went to bed for three weeks, during which time he still had some pain. He then returned to work, but after two weeks he noticed that he was very short of breath; he had little if any epigastric pain. He had been admitted to hospital six weeks before the time of the meeting, and on examination was dyspnœic at rest. His blood pressure was 170 millimetres of mercury, systolic, and 150 millimetres, diastolic; his heart was clinically enlarged with a systolic murmur audible at the apex and a duplicated second pulmonary sound. The liver was palpable two fingers' breadth below the costal margin, and there was slight œdema of the ankles. The rest of the examination yielded normal findings. X-ray examination showed an enlarged heart (both to right and to left) with small effusions at both lung bases. The blood sedimentation rate and the results of X-ray examination after a barium meal, and of Wassermann and urea concentration tests were all normal. The electrocardiogram showed tracings of poor amplitude with absence of *T* waves in all leads; some left axis deviation was also noted. Treatment with bed rest, "Digoxin", aminophyllin and a diet of high protein content had so far caused no improvement; the patient still had periods of dyspnœa and on occasions had a certain amount of albuminuria. Dr. Farrar presented the patient as an example of a sufferer from an unusual form of cardiac failure.

Chronic Hepatitis and Splenomegaly.

DR. W. ROSENTHAL then showed a patient, a single girl, aged twenty-one years, who had given a history of being well until the age of ten years, when she commenced to suffer from numerous "blackouts", which were sometimes associated with vomiting. Those attacks had now ceased. Three years previously she had become jaundiced and was admitted to the Royal Melbourne Hospital; a diagnosis of chronic hepatitis was made, this being supported by a liver biopsy. The patient's symptoms subsided, and she was well for one year. Two years before the meeting she had commenced to suffer from recurrent severe attacks of pain under her left costal margin, and this radiated down her left side. She was treated at home for the attacks of pain, which eventually became so persistent that they resulted in her being sent down to the Bendigo Base Hospital. On admission to hospital, the patient was found to have a slight icteric tinge, and on palpation of her abdomen an enlarged tender spleen was detected. No other abnormality was found on examination. The attacks of pain at times required injections of morphine for relief. Their onset was sudden, and they persisted for several hours. At times the pain appeared to be controlled by injections of sterile water and was often referred to the left shoulder tip. The attacks were often associated with constipation. After several weeks' bed rest, the attacks had become less frequent and the patient was returned home. However, the incidence of the attacks again increased, and she was readmitted to hospital with the diagnosis of chronic hepatitis and splenomegaly. It was considered that the chronic hepatitis had resulted in an increased pressure in the portal system with splenic engorgement, and that the attacks of pain might have been associated with perisplenitis and splenic infarction. Further investigations revealed a total leucocyte count of 4500 per cubic millimetre with a monocytosis of 31%. A cholecystogram revealed a non-functioning gall-bladder. Excretion pyelography yielded normal findings. The result of a Casoni test was negative. The serum bilirubin value was 2·8 milligrammes per 100 millilitres, and the serum protein value was 6·8 grammes *per centum*, of which 3·8 grammes *per centum* were albumin. Dr. Rosenthal explained that at the present time the question of splenectomy was being considered.

Cystic Fibroid Tumour Complicating Pregnancy.

DR. E. SANDNER discussed a case of a cystic fibroid complicating pregnancy. The patient, a woman, aged thirty-six years, had been first examined seven months previously, when she complained of frequency of micturition, gross loss of weight and lower abdominal and lower back pain. Pelvic examination revealed a cystic mass filling the pelvis, with displacement of the cervix forward and to the left, and small rounded tumours palpable on what was thought to be the anterior wall of the uterus. The result of an Aschheim-Zondek test was positive. Five months later the patient was admitted to hospital with the cystic swelling now palpable to the level of the umbilicus and a solid mass about half-way between the umbilicus and pubis. She said that she had missed her last two menstrual periods. X-ray examination failed to show any fœtal parts, and operation was performed. This revealed a large subperitoneal cystic mass, with a small solid mass on its anterior wall, and a small uterus. The firm attachment of the cyst to the posterior wall of the uterus necessitated total hysterectomy for its removal. The following pathological report was given on the tumour:

> Both tumours, the smaller as well as the larger one, are basically fibromyomata. They have first of all undergone very extensive hyaline degeneration, which in the microscopic sections produces a most intricate pattern, while in the larger tumour the degenerative changes have progressed through mucoid degeneration to cyst formation. Although the tumours are, in the not degenerated parts, quite cellular, there is no evidence of malignancy.

Supracondylar Fracture of the Humerus.

Dr. Sandner also showed a series of patients with supracondylar fracture of the humerus and discussed methods of reduction.

Spontaneous Fracture of the First Rib.

DR. E. BIGGS showed a patient who had had spontaneous fracture of the first rib on the right side, confirmed by X-ray examination as being at about the position of the scalene tubercle. After seven weeks, X-ray examination showed no signs of union or callus formation. The patient was a man, aged twenty-five years; symptoms of a tight feeling in the chest at the area of the angle of the sternum, with soreness on pressure in this area, had followed an attack of influenza about four months before.

Cyst in the Common Bile Duct.

DR. A. L. NEWSON showed a girl, aged twelve years, who had been first examined about one year previously, when she had a history of jaundice for eight months, slight abdominal discomfort, and progressive and severe loss of weight (four stone). Her past history included *melæna neonatorum* and a perforated appendix which had been removed at the age of five years. On examination the child was found to be deeply jaundiced, and abdominal palpation revealed a large, rounded, cystic tumour occupying the right half of the abdominal cavity. It moved downwards on inspiration and was slightly tender. In size it could be compared to a "soccer" football. Investigations revealed bile in the urine, a slight degree of anæmia, and negative results from a Casoni test and chest X-ray examination. She was given an injection of vitamin *K* and a blood transfusion, and at operation the cyst was found to be in part of the common bile duct; it was possible to trace it upwards to the common hepatic duct and to the cystic duct. The duodenum was contiguous with the medial wall of the cyst, but entrance into the former could not be demonstrated. An anastomosis of the wall of the cyst to the anterior surface of the pyloric antrum was carried out, but the post-operative course was stormy with pyloric obstruction becoming evident. Therefore a second operation was carried out one month later. The cyst was found to have shrunk to the size of a tennis ball and to be firmly attached to the pylorus; to undo this anastomosis appeared too risky, so a posterior gastro-jejunostomy was quickly carried out. After that there was no further evidence of obstruction, but the child developed a high temperature, deepening jaundice and pain, and tenderness over the liver about two weeks later. Streptomycin appeared to be the deciding factor in clearing up this complication, and thereafter progress was rapid, with disappearance of the jaundice and increased weight gain. At the time of the meeting the girl appeared perfectly well.

Traumatic Rupture of Transverse Colon.

Dr. Newson's second patient was a boy, aged three years, who had been admitted to hospital with a history of having been kicked in the abdomen by a horse half an hour previously. There were no external marks of violence, but the abdomen was rigid and very tender, especially in the left upper quadrant. An immediate operation was carried out, and the peritoneal cavity was found to be full of blood-stained fæculent fluid. A ragged tear, three-quarters of an inch in diameter, was found in the transverse colon, and this damaged segment was exteriorized as a loop colostomy after the fæculent peritoneal contents had been sucked out.

A blood transfusion and intravenous infusion of fluid were given post-operatively. Progress was uneventful, and the colostomy opening was closed one month later. The child was extremely well.

Paraduodenal Hernia.

Dr. Newson then showed a girl, aged thirteen years, who had been admitted to hospital with a history of central colicky abdominal pain, vomiting and constipation for one day. She had apparently had similar attacks for six years. The lower part of the abdomen was found to be tender but not rigid. At operation a paraduodenal hernia was found, containing a large amount of small intestine, which, when delivered, was found to be quite undamaged. Convalescence in this case was quite uneventful.

Correspondence.

AN ADDRESS.

SIR: I read with interest Dr. Rawle's address given to the annual meeting of the New South Wales Branch of the British Medical Association. Dr. Rawle has summarized a very wide and difficult subject extremely well.

There are some parts of this which call for comment. The establishment of a committee to make a reappraisal of general practice would serve a very useful and worthwhile object, if only realigning the general practitioners in the suburbs of the metropolitan area with the general hospitals, from which in many cases they are excluded.

Two other points which are of the utmost importance. Firstly, reduction, in some measure at least, of the large amount of clerical work devolving upon the general practitioner and eating heavily into his time. Secondly, the question of some special arrangement with regard to "locums" is another extremely "sore" point with general practitioners. The average young graduate, fresh off the press of the medical school, is not properly equipped to handle a general practice. As Dr. Rawle rightly points out, he has been instructed by specialists in established diseases and knows little or nothing about incipient diseases, which form nearly three-quarters of a general practice.

On the business side of practices, nearly all new graduates are completely ignorant of this, and in my own protection, I have drawn up a set of guiding instructions, which I put in the hands of the "locum" when I am briefing him.

Another excellent suggestion made by Dr. Rawle which the authorities might well bear in mind is that new graduates should be assigned to a well-established general practitioner for a period of six months. This would serve two purposes and would assist the general practitioner in some of his work, and in return the new graduate would be taught something of the conduct of medical practices. Every general practitioner in the course of time and out of his own experience evolves methods of book-keeping and of conducting his practice—frequently by means of trial and error method. Much good could possibly come from an investigation by a committee such as Dr. Rawle suggests. This committee could conceivably, out of the information placed in their hands from the general practitioners, evolve a well worthwhile method which could be published in booklet form and which would, I am sure, prove of inestimable value to new practitioners. As a young practitioner, I am sure that I and many others would have welcomed such guidance.

Yours, etc.,

Centennial Park, K. J. B. DAVIS.
New South Wales,
May 29, 1951.

THE PRESCRIPTION OF LIFE-SAVING DRUGS.

SIR: Why have we had no editorial protest against the latest iniquitous system of prescribing life-saving drugs—"Chloromycetin", streptomycin *et cetera*.

Perhaps it is all right in the city, where a telephone call settles the problem, but in the country we have to contend with telegraph offices being closed, and cannot discuss the case with the appropriate official, who can only blindly say yes or no, thus diagnosing a case on one or two words at a distance of hundreds of miles. And is it not a flagrant breach of professional secrecy to put a patient's name and diagnosis on a telegram? Are we to lose our professional conscience without even a struggle?

Who pays for these telegrams? Do I? Or do I charge the patient, and if so, is this "free" medicine?

The circular states that this system is in operation because these drugs are in short supply—it arrived about three days after a circular from the manufacturers of "Chloromycetin", stating it was available in unlimited supply.

The amount available on each telegram is up to the maximum under the *Pharmaceutical Benefits Act*; thus, if a patient is to have one gramme of streptomycin daily for thirty days, it means sixty telegrams.

In conclusion, it is the really life-saving drugs which are required more urgently which are subjected to delay, and it is in the country where diagnostic facilities are not as good which has the more difficult job of obtaining these steam-hammer drugs.

Surely the system can be changed.

Yours, etc.,

Ayr, ROBERT N. NELSON.
North Queensland,
May 31, 1951.

"ADDICTS ANONYMOUS."

SIR: A few patients who have been cured of drug addiction by the methods of Alcoholics Anonymous are anxious to form a new group, "Addicts Anonymous", in Australia. The group takes its name from similar groups who have worked successfully in America.

The groups depend entirely on the Alcoholics Anonymous programme, and are in no way dissociated from that body. They feel, however, that as the problem of drug addicts varies somewhat from those of alcoholics, addicts can be best helped by those who had once been addicts themselves. All that is demanded of new members is that they have a sincere and honest desire to give up taking drugs.

The new body can be communicated with at UM 8537.

Yours, etc.,

195 Macquarie Street, S. J. MINOGUE.
Sydney,
May 31, 1951.

PENETRATING INJURIES OF THE RECTUM.

SIR: In your issue of May 5, 1951, Dr. Edward Wilson reports four interesting cases of injuries to the rectum, and discusses the treatment in general for such cases, and the operative procedure carried out by him.

Twenty-five years ago, soon after starting practice, I operated on a man, thirty years of age (in a country town), eleven hours after he had jumped off a wagon onto a pitchfork standing up with its prongs in the ground. The handle had penetrated the anus, perforated the front of the rectum, penetrated the bottom of the bladder, perforated the top of the bladder, injured the mesoappendix and partially torn off the appendix, and had come to rest (it was estimated) up under the liver, at which juncture the man's feet had reached the ground and stopped further progress of the handle.

It may interest Dr. Wilson, and others, to know just what was done, and, perhaps more particularly, what was not done, as the man completely recovered without any disability at all.

Through a suprapubic mid-line incision the injured appendix was removed; the top of the bladder was repaired with three layers of sutures. Nothing was done to the base of the bladder or to the rectum. The abdominal incision was closed without drainage. A catheter was secured in the bladder through the penis and left in for three days. Twelve hours after the operation the patient was given half-grain doses of calomel every half-hour until eight grains had been given. This was followed by three one-drachm doses of liquid extract of cascara at hourly intervals, which resulted in the bowels being opened. The patient was given hexamine throughout (grains seven and a half three times a day for three days) and a potassium citrate mixture every fourth day. The patient left hospital in seven weeks, quite well.

This man is alive and well today.

This case was reported by me in detail in THE MEDICAL JOURNAL OF AUSTRALIA of December 6, 1924.

Yours, etc.,

Burwood, WALTER J. HULL.
New South Wales,
May 9, 1951.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course for Diploma in Anæsthesia.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the course for the diploma in anæsthesia, proposed to begin on June 18, 1951, will not begin until September 3, when the Part I lectures will commence. Intending candidates may obtain details on application to the Course Secretary of the Committee, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. The closing date for applications will be August 13, 1951.

POST-GRADUATE COMMITTEE IN MEDICINE OF THE UNIVERSITY OF ADELAIDE.

Course Suitable for Candidates for F.R.A.C.S. Part I.

A THREE-MONTH FULL-TIME COURSE suitable for candidates for F.R.A.C.S. Part I, including lectures in anatomy, applied physiology and the principles of pathology, lecture-demonstrations in histology, vivas and test questions, will begin on Monday, July 9, 1951. The fee will be £31 10s. Applications, accompanied by cheques, should be sent to the Secretary not later than July 2, 1951.

Refresher Week and Week-End Course.

The refresher week will be held from August 20 to 24, 1951, inclusive, and will consist of lectures, demonstrations and ward rounds in medicine, surgery, obstetrics, pædiatrics and the specialties. Professor D. M. Dunlop, the Sims Travelling Professor, and Professor Lorimer Dods, the Edward Stirling Lecturer, will both take part in the course. A detailed syllabus may be obtained from the Secretary. The fee will be £3 3s.

The subject of the week-end course will be "Road Accidents". The course will be held on August 25 and 26, 1951, and the fee will be £3 3s. A detailed syllabus may be obtained from the Secretary.

The inclusive fee for refresher week and week-end course will be £5 5s. Applications for each or both, accompanied by cheques, should be sent to the Secretary not later than August 13, 1951.

SEMINARS AT ROYAL PRINCE ALFRED HOSPITAL.

THE following seminars will be held at Royal Prince Alfred Hospital, Camperdown, New South Wales, between June 1 and October 26, 1951. Each seminar is held on Friday from 1.15 to 2.15 p.m. in the A2 lecture theatre. All medical graduates are invited to attend.

June 22: Gastro-enterology Section, "Therapeutics" (Professor D. M. Dunlop).
June 29: Thoracic Section, "Atypical Pneumonia".
July 6: Neurology Section, "Cauda Equina Lesions".
July 13: Pædiatrics Section, "Rheumatism in Childhood".
July 20: Cardio-vascular Section, "New Hypertensive Drugs".
July 27: Clinico-pathological conference.
August 3. Surgical seminar (Royal Prince Alfred Hospital reunion).
August 10: No seminar.
August 17: Endocrine and Metabolism Section, "Medical Complications of Pregnancy".
August 24: Hæmatology Section, "Intravascular Hæmolysis".
August 31: Thoracic Section, Journal Club.
September 7: Gastro-enterology Section, "Gastric Cancer".
September 14: Pædiatrics Section, "Anæmias in Childhood".
September 21: Neurology Section, "Experiences with Subdural Hæmatoma".
September 28: Cardiovascular Section (Dr. Paul Wood, London).
October 5: Endocrinology and Metabolism Section, "Potassium Intoxication".
October 12: Hæmatology Section, Journal Club.
October 19: Thoracic Section, "Impressions Abroad" (Dr. Maurice Joseph).
October 26: Gastro-enterology Section, Journal Club.

University Intelligence.

UNIVERSITY OF MELBOURNE.

THE following information is taken from the *University of Melbourne Gazette* of May 4, 1951:

Professor R. D. Wright has been elected unopposed as a representative of the professors on the University Council. He succeeds Emeritus Professor P. MacCallum.

The University Council has approved the following new appointments: Dr. G. S. Christie, Senior Lecturer in Pathology; Dr. H. Eddey, Independent Lecturer in Surgical Anatomy; Dr. F. X. G. Hurley, Clinical Supervisor at Saint Vincent's Hospital.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, promotions *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 33, of May 18, 1951.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointments.—Edward Winston Freshney (Surgeon Lieutenant-Commander, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant-Commander, with seniority in rank of 12th December, 1948, dated 13th February, 1951. Edgar John Hardcastle (Surgeon Lieutenant, Royal Australian Naval Volunteer Reserve) is appointed Surgeon Lieutenant, with seniority in rank of 26th August, 1945, dated 13th February, 1951. John Damien Tange is appointed Surgeon Lieutenant, dated 12th February, 1951.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps (Medical).

NX208064 Captain (Temporary Major) D. C. Cook ceases to be seconded to the Department of Supply, 15th February, 1951.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (Provisionally), 23rd February, 1951.—1/39103 Edward Gilles Ahern.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/101005 Major (provisionally) T. D. Hollywood is seconded for post-graduate studies in the United Kingdom, 10th January, 1951.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/35230 Lieutenant-Colonel R. T. Binns, O.B.E., is appointed from the Reserve of Officers and is borne supernumerary to the authorized establishment of Lieutenant-Colonels, with pay and allowances of Major (at his own request), 22nd January, 1951. The provisional ranks of the following officers are confirmed: 4/35217 Major F. B. Turner, Captains 4/31955 H. R. Moore, 4/31963 V. D. Plueckhahn and 4/31965 R. R. A. Brock.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

1st Military District: To be Honorary Captain, 13th March, 1951.—Roger Alwin Bennett.

3rd Military District.—The resignation of Captain G. P. Cromie of his commission is accepted, 28th February, 1951. To be Honorary Captain, 7th March, 1951: James David Clancy.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The grant of special leave without pay to Flight Lieutenant J. N. Diggle (033066), as approved in Executive Council Minute No. 3 of 1951, appearing in *Gazette* No. 7, dated the 25th January, 1951, is amended to read from the 8th August, 1950, to the 7th March, 1951, inclusive.

Dr. Bryan Hartley, M.R.C.S., L.R.C.P. (034368) is appointed to a short-service commission, on probation for a period of twelve months, 17th January, 1951, with the rank of Flight Lieutenant.

Air Force Reserve.

Medical Branch.

The appointments of the following officers are terminated: Temporary Squadron Leader S. W. Dobell-Drown (261658), 18th December, 1950, Flight Lieutenant J. Barnes (264590), 8th November, 1950.

The following are appointed to commissions with the rank of Flight Lieutenant: John Armstrong Snell (257806), 9th February, 1951, Harris Neville Greenberg (267907), 13th February, 1951.

Notice.

COLLEGE OF RADIOLOGISTS (AUST. AND N.Z.).

THE College of Radiologists (Aust. and N.Z.) invites all members of the medical profession to attend a lecture to be given by Professor B. W. Windeyer on Tuesday, June 19, 1951, at 8 p.m. in the Stawell Hall, Macquarie Street, Sydney. There will be no charge. Professor Windeyer will speak on carcinoma of the larynx (illustrated with a sound film) and carcinoma of the tongue (illustrated with slides).

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1945, as duly qualified medical practitioners:

Ackary, John Francis, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Anderson, Gerard James, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Anderson, Hugh Cairns, M.B., B.S., 1951 (Univ. Sydney), District Hospital, Wellington.

Andrea, Brian Maxwell, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Atkinson, Kenneth John James, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Bailey, Harry Richard, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Bannon, Paul Edward, M.B., B.S., 1951 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Barbour, Edmund, M.B., B.S., 1951 (Univ. Sydney), District Hospital, Marrickville.

Bearup, Dorothy Joy, M.B., B.S., 1951 (Univ. Sydney), Rachel Forster Hospital, Redfern.

Bell, Peter Kenneth, M.B., B.S., 1951 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Birbara, George, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Blunt, John Littleton, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.

Bowman, Reginald, M.B., B.S., 1951 (Univ. Sydney), St. George Hospital, Kogarah.

Brassil, John Vincent Carlin, M.B., B.S., 1951 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Brassil, Joseph Alexis Carlin, M.B., B.S., 1951 (Univ. Sydney), Saint Joseph's Hospital, Auburn.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 19, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[2]
Ankylostomiasis	*	..	2	..	1	..	..	..	3
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	2(2)	4(3)	..	..	..	..	..	..	6
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	*	*	*	*	*	*	..
Dengue	*	*	..	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	1(1)	..	2(1)	..	..	..	3
Diphtheria	17(7)	7(1)	8(3)	..	13(9)	..	..	..	45
Dysentery (Amœbic)	*	..	..	..	1	..	..	..	1
Dysentery (Bacillary)	*	1(1)	13(13)	..	..	..	..	2	16
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	1	*	*	..	..	1
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	3	*	*	..	*	*	..	3
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	12(9)	*	*	..	..	12
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	30(16)	7(3)	36(11)	22(17)	..	..	..	1	96
Psittacosis	*	*	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*c*)	*	*	..	*	2	*	..	..	2
Scarlet Fever	24(13)	26(18)	6(1)	10(6)	3(1)	7(1)	..	..	76
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	34(28)	24(21)	7(5)	6(5)	9(8)	6(1)	..	..	86
Typhoid Fever(*e*)	..	..	..	..	1	..	..	..	1
Typhus (Endemic)(*f*)	..	..	2	..	2	..	..	..	4
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	*	*	*	..	2	2
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946-1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Brown, Trevor John Gyton, M.B., B.S., 1951 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest.
Brustolin, Nerino, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Sydney.
Bull, Richard Stuart, M.B., B.S., 1951 (Univ. Sydney), 491 New South Head Road, Double Bay.
Burfitt-Williams, Walter John, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.
Callaghan, Ronald Alan, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Campion, Frederick John, M.B., B.S., 1951 (Univ. Sydney), Saint Joseph's Hospital, Auburn.
Carrick, Denise Myles, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Caspersonn, Douglas James, M.B., B.S., 1951 (Univ. Sydney), District Hospital, Balmain.
Cass, Moses Henry, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Castledon, Rosalind Archer, M.B., B.S., 1951 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.
Chalmers, Robert, M.B., B.S., 1951 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.
Chambers, Ross Campbell, M.B., B.S., 1951 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Channells, Raymond Robert, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Collins, John James, M.B., B.S., 1951 (Univ. Sydney), Lewisham Hospital, Lewisham.

Honours.

BIRTHDAY HONOURS.

INCLUDED among those who have received Birthday Honours from His Majesty the King is Dr. Philip Santo Messent, of Adelaide, who has been created a Knight Bachelor.

Obituary.

PEDER NIELSEN PEDERSEN.

WE regret to announce the death of Dr. Peder Nielsen Pedersen, which occurred on June 4, 1951, at Edinburgh, Scotland.

AINSLIE FRANCIS AGNEW.

WE regret to announce the death of Dr. Ainslie Francis Agnew, which occurred on June 4, 1951, at Melbourne.

Medical Appointments.

Dr. R. E. Wood has been appointed Public Vaccinator for the Shire of Bright, Victoria.

Dr. A. H. M. Maxwell has been appointed Public Vaccinator for the Shire of Violet Town, Victoria.

Dr. J. M. Sleeman has been appointed Public Vaccinator for the Shire of Wycheproof, Victoria.

Dr. M. Fowler has been appointed Public Vaccinator for the Shire of Bulla, Victoria.

Dr. J. E. Hughes has been appointed honorary assistant surgeon at the Royal Adelaide Hospital.

Dr. E. B. Jones has been appointed President of the Medical Board of South Australia.

Dr. R. J. S. Walker has been appointed honorary assistant radiologist at the Royal Adelaide Hospital.

Dr. D. H. Cohen has been appointed honorary assistant surgeon at the Royal Alexandra Hospital for Children, Camperdown, Sydney.

Dr. J. B. Dowe has been appointed honorary assistant ear, nose and throat surgeon at the Royal Alexandra Hospital for Children, Camperdown, Sydney.

Dr. D. S. Brandt has been appointed honorary assistant physician in charge of psychiatric and child guidance clinic at the Royal Alexandra Hospital for Children, Camperdown, Sydney.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Goodman, Harry Thomas, M.B., B.S., 1950 (Univ. Sydney), Haylock Street, Tullamore.
Watson, Lyal Clifton Albert, M.B., B.S., 1950 (Univ. Sydney), 20 Tryon Road, Lindfield.
Dobell-Brown, David Leslie, M.B., B.S., 1951 (Univ. Sydney), The Royal North Shore Hospital, St. Leonards.
Croucher, Colin, M.B., B.S., 1950 (Univ. Adelaide), Box 277, P.O., Broken Hill.

Diary for the Month.

JUNE 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 20.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 21.—South Australian Branch, B.M.A.: Clinical Meeting.
JUNE 21.—Victorian Branch, B.M.A.: Executive Meeting.
JUNE 22.—Queensland Branch, B.M.A.: Council Meeting.
JUNE 26.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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